



ORAL & MAXILLOFACIAL SURGERY REVIEW

A STUDY GUIDE

Edited by
Din Lam, DMD, MD
Daniel M. Laskin, DDS, MS

Oral and Maxillofacial Surgery Review: A Study Guide

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Contents



Preface v

Contributors vi

1 Medical Assessment 1
Alia Koch and Steven M. Roser

2 Anesthesia 49
Jason Jamali and Stuart Lieblich

3 Dentoalveolar Surgery 75
Esther S. Oh and George Blakey

4 Dental Implantology 119
Christopher Choi and Daniel Spagnoli

5 Orthognathic Surgery 135
David Alfi and Jaime Gateno

6 Trauma 159
Daniel E. Perez and Edward Ellis III



7 Pathology 195
Din Lam and Eric R. Carlson

8 Maxillofacial Reconstruction 275
Din Lam and Andrew Salama

9 Orofacial Pain 317
David W. Lui and Daniel M. Laskin

10 The TMJ 331
David W. Lui and Daniel M. Laskin

11 Craniofacial Surgery 363
Jennifer Woerner and Ghali E. Ghali

12 Cosmetic Surgery 391
David E. Webb and Peter D. Waite

Index 421

Preface

When faced with a board or recertification examination, or just wanting to update your knowledge in oral and maxillofacial surgery, there is always the dilemma of where to begin and what and how much to study. This review book is designed to help you with this process. The format involves a detailed outline of the important items of didactic and clinical information in the 12 major areas of oral and maxillofacial surgery combined with numerous tables, summary charts, and useful mnemonics and surgical tips. Each chapter is also supplemented with many clinical photographs, diagrams, and photomicrographs, where appropriate.

The various chapters were each developed by two authors: (1) a young oral and maxillofacial surgeon who was board certified in recent years and is therefore very familiar with the process and the content, and (2) a more senior surgeon who has been a board examiner and/or involved in the recertification process and is therefore knowledgeable and experienced in the essential clinical aspects of the specialty.

To use this book as a review or study guide, it is suggested that you first read each chapter to determine what information is already familiar to you, what is new and needs to be learned, and what are the areas in which you desire more information from other sources such as the list of recommended reading at the end of each chapter. The last material should then be annotated where indicated in the various chapters. The book now becomes your study manual as well as a quick and easy way to review the material again just prior to the examination and a handy reference source during clinical practice.

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Medical Assessment

Alia Koch and Steven M. Roser

- ▶ Cardiovascular Disease
- ▶ Respiratory Disease
- ▶ Renal Disease
- ▶ Acid-Base Disorders
- ▶ Gastrointestinal Disease
- ▶ Hematologic Disease
- ▶ Endocrine Disease
- ▶ Autoimmune Disease
- ▶ Neurologic Disorders
- ▶ Perioperative Management

Cardiovascular Disease

Acute Coronary Syndrome

Major blood vessels supplying the heart are damaged/diseased by cholesterol plaques, which cause the vessels to narrow. In turn, less blood reaches the myocardium, leading to an acute coronary syndrome.

- **Symptoms:** Dull substernal pain and pain radiating to left arm and jaw; associated with diaphoresis, dyspnea
- **Diagnosis:** electrocardiogram (ECG), cardiac enzymes
 - ST segment elevation myocardial infarction (STEMI)
 - Treatment: Immediate reperfusion (angioplasty or thrombolytic therapy) within 12 hours of onset of chest pain
 - Non-ST segment elevation myocardial infarction (NSTEMI)
 - Treatment: Medical therapy (aspirin, beta blockade, angiotensin-converting enzyme [ACE] inhibitor)
 - Unstable angina
 - Treatment: Medical therapy (same as NSTEMI)

Congestive Heart Failure

- Systolic heart failure: Reduced ejection fraction ($< 40\%$), S3 murmur, dilated left ventricle
- Diastolic heart failure: Preserved ejection fraction ($> 50\%$), S4 murmur, left ventricle hypertrophy
- **Symptoms:** Chest pain, shortness of breath, orthopnea, extremity swelling, jugular vein distention
- **Diagnosis**
 - Echocardiogram: Evaluate heart motion, ejection fraction
 - ECG: Evaluate changes in ECG, heart strain
 - Stress test: Evaluate coronary artery disease
 - Brain natriuretic peptide: Normal value rules out acute heart failure
 - Chest radiograph: Evaluate heart size, fluid in the intrathoracic cavity

Classification of congestive heart failure (CHF)

Stage	Definition	Treatment
A	Risk of HF due to comorbidities only	Treat underlying condition
B	No symptoms but structural abnormality predisposes patient to HF	ACE inhibitor, beta blocker
C	Structural disease with HF symptoms	ACE inhibitor, beta blocker, diuretic, salt restriction
D	HF symptoms at rest	Medical therapy with mechanical support

HF, heart failure.

Valvular Disease

	Symptoms	Murmur	Diagnosis	Treatment
Aortic stenosis	Angina, syncope, HF	Crescendo-decrescendo systolic murmur	Echocardiogram, with severity determined by valve area and mean gradient	Aortic valve replacement for symptomatic patients
Aortic regurgitation	Progressive dyspnea on exertion, with signs of HF	Decrescendo blowing diastolic murmur	Echocardiogram	<ul style="list-style-type: none">• Afterload reduction with systemic vasodilators and diuretics• Valve replacement in worsening cases
Mitral stenosis	Gradual onset with dyspnea on exertion, right HF, and pulmonary hypertension	Opening snap	Echocardiogram with severity determined by valve area and transmitral pressure gradient; atrial fibrillation often present	Medical therapy, valvuloplasty, mitral valve replacement
Mitral regurgitation	Asymptomatic increasing to dyspnea on exertion and HF	Holosystolic, blowing murmur	Echocardiogram	<ul style="list-style-type: none">• If ejection fraction < 30%, valve replacement• If ejection fraction > 30%, medical therapy, but if resistant, left ventricular assist device
Mitral valve prolapse	Asymptomatic	Midsystolic click	Echocardiogram	None if asymptomatic
Mitral valve prolapse syndrome	Chest pain, palpitations, anxiety, skin tingling, syncope	Midsystolic click	<ul style="list-style-type: none">• Supraventricular tachycardia• Autonomic nervous system dysfunction	Reassurance, lifestyle changes, stress reduction
HF, heart failure.				

Arrhythmias

Pattern	Etiology	Symptoms	Diagnosis	Treatment
Ventricular tachycardia	Myocardial infarction, cardiomyopathy, electrolyte abnormalities, blunt trauma, infectious or infiltrative disease	Chest pain, dyspnea, syncope	<ul style="list-style-type: none">• Monomorphic: Uniform QRS pattern (scar)• Polymorphic: Varied QRS (torsades)	Cardioversion

(Arrhythmias cont)

Pattern	Etiology	Symptoms	Diagnosis	Treatment
Atrial fibrillation	Hypertension, valvular disease, coronary artery disease, HF	Palpitations, fatigue, dyspnea, dizziness	Absent P waves	<ul style="list-style-type: none">• Unstable: Cardioversion• Stable: Rate control, rhythm control, use CHADS₂ criteria for anticoagulation therapy (see below)
Atrial flutter	Reentry circuit in the right atrium	Asymptomatic to palpitations, decreasing exercise tolerance, dyspnea	Continuous, regular atrial activity with sawtooth pattern	<ul style="list-style-type: none">• Unstable: Cardioversion• Stable: Antiarrhythmics with consideration for ablation if long term
Paroxysmal supraventricular tachycardia (PSVT)	Atrioventricular node reentry and ectopic atrial foci	Palpitations, lightheadedness, chest discomfort	ECG, Holter monitor	Vagal maneuvers, adenosine, medical management, ablation
Wolff-Parkinson-White (WPW) syndrome	Accessory pathway between atria and ventricles due to congenital separation during fetal development; risk of sudden cardiac death and tachyarrhythmias	Palpitations, lightheadedness, loss of consciousness	Delta waves on ECG	Catheter (radiofrequency) ablation
Bradycardia	Ischemic, infectious, infiltrative, autoimmune, conditioned heart, medication, metabolic, neurologic	Dizziness, weakness, fatigue, HF, loss of consciousness	ECG, tilt table	<ul style="list-style-type: none">• Unstable: ACLS• Stable: Treat underlying cause, atropine, pacing

HF, heart failure; ACLS, advanced cardiovascular life support.

CHADS₂ scoring table

Stroke risk assessment in atrial fibrillation to determine necessity of anticoagulation or antiplatelet treatment.

	Condition	Points
C	Congestive heart failure	1
H	Hypertension: Blood pressure consistently above 140/90 mm Hg (or treated hypertension with medication)	1
A	Age ≥ 75 years	1
D	Diabetes mellitus	1
S₂	Prior stroke or transient ischemic attack (TIA) or thromboembolism	2

Stroke risk assessment

Score	Risk	Treatment
0	Low	Aspirin or none
1	Moderate	Aspirin or coumadin to INR of 2-3
2 or more	Moderate/high	Coumadin to INR of 2 to 3

INR, international normalized ratio.

Heart block (Fig 1-1)

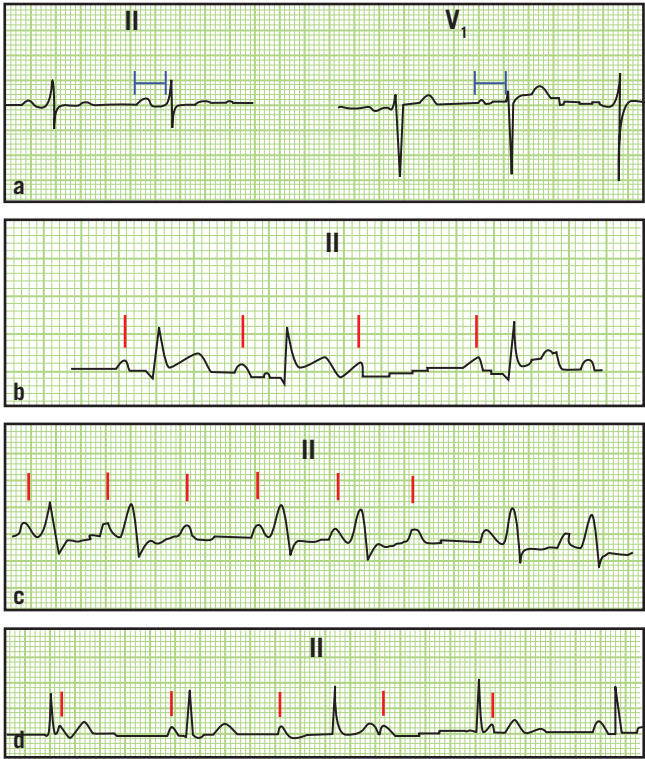


Fig 1-1 Heart block ECG strips. (a) First degree. (b) Second degree, type 1. (c) Second degree, type 2. (d) Third degree. P waves indicated by a red vertical line. (Reprinted with permission from EKG-Uptodate 2013.)

	ECG finding	Treatment
Type 1	Increased PR Interval	None
Type 2A	Increasing PR interval until dropped QRS	Pacemaker for symptomatic patients only
Type 2B	Regularly dropped QRS with constant PR interval	Search for cause/pacemaker
Type 3	Complete dissociation of P waves and QRS complexes	Search for cause/pacemaker

Hypertension

- Primary hypertension: No identifiable cause
- Secondary hypertension: Identifiable cause, some listed below
 - Renal artery stenosis – Diabetic nephropathy – Thyroid disease – Cocaine use – Pheochromocytoma
 - Obstructive sleep apnea
- **Diagnosis:** At least two elevated BP readings on at least two different occasions
 - Prehypertension: Systolic blood pressure (SBP) from 120 to 130 mm Hg, diastolic blood pressure (DBP) from 80 to 89 mm Hg
 - Stage 1: SBP from 140 to 159 mm Hg, DBP from 90 to 99 mm Hg
 - Stage 2: SBP \geq 160 mm Hg, DBP \geq 100 mm Hg
- **Etiology:** Obesity, familial, smoking, diabetes, kidney disease, Cushing syndrome, catecholamines, obstructive sleep apnea
- **Treatment:** Diet, weight reduction, aerobic activity, sodium restriction, medications

Medications

	Indication	Side effect	Examples
Beta blocker	MI, CAD risk, CHF	Bronchospasm, atrioventricular node blockade	Atenolol, metoprolol
ACE inhibitor	Diabetes, MI, proteinuria, CHF	Cough, renal failure	Enalapril, captopril
Angiotensin receptor blocker	Patients who cannot tolerate ACE inhibitor	Renal failure	Valsartan, losartan
Thiazide	Combination therapy	Hypokalemia	Hydrochlorothiazide
Calcium channel blocker	Systolic hypertension, CAD	Conduction blockade	Diltiazem, verapamil

CAD, coronary artery disease; MI, myocardial infarction.

Hypertensive emergencies

	Blood pressure	Findings	Treatment
Urgent	> 180/120 mm Hg	No end organ damage	Medical therapy on outpatient basis
Emergency	> 180/120 mm Hg	Evidence of end organ damage	Admission with IV therapy
IV, intravenous.			

Infective Endocarditis (IE)

Type	Cause
IV drug use	<i>Staphylococcus aureus</i>
Native valve	Viridans streptococci, <i>S aureus</i> , enterococci
Prosthetic valve	<i>Staphylococcus epidermidis</i> , <i>S aureus</i>
Culture negative	HACEK organism (<i>Haemophilus</i> , <i>Aggregatibacter</i> , <i>Cardiobacterium</i> , <i>Eikenella corrodens</i> , <i>Kingella</i>), <i>Candida</i> , <i>Aspergillus</i>
IV, intravenous.	

Duke criteria for diagnosis

- Definite IE: 2 major; 1 major and 3 minor; 5 minor
- Possible IE: 1 major and 1 minor; 3 minor

Major criteria	Minor criteria
<ul style="list-style-type: none">Positive blood cultureEchocardiogram with evidence of endocardial involvement	<ul style="list-style-type: none">Predisposition to IE (IV drug use, indwelling catheter, diabetes)FeverVascular phenomena (Janeway lesions, arterial emboli, intracranial hemorrhage, splinter hemorrhage)Microbiologic evidenceImmunologic phenomena (Osler nodes, Roth spots)
IV, intravenous.	

Treatment

- Native valve endocarditis: Vancomycin and gentamicin
- Prosthetic valve endocarditis: Vancomycin, rifampin, and gentamicin
- Culture positive: Treat organism

Surgical Management of Patients on Cardiovascular Medications

Preoperative treatment decision algorithm

1. Urgent surgery, nonurgent surgery with unstable/active cardiac condition
 - Medical consult/discussion with surgeon
2. Nonurgent surgery
 - Surgical procedure risk (Box 1-1)
 - Low risk: Medical consult preoperatively
 - Moderate/high risk: Go to step 3
3. Evaluate patient's functional capacity (Box 1-2)
 - > 4 METs: Statin therapy and beta blocker preoperatively
 - ≤ 4 METs: Go to step 4
4. Evaluate risk of surgical procedure (see Box 1-1)
 - Moderate risk: Statin, beta blocker, ECG, possible ACE inhibitor
 - High risk: Go to step 5
5. Evaluate cardiac risk factors (Box 1-3)
 - ≤ 2: Preoperative statin, beta blocker, possible ACE inhibitor
 - > 2: Noninvasive testing, discuss anesthesia technique, consider changing surgical management

MET, metabolic equivalent of task.

Box 1-1 Risk of surgical procedures

Low risk	Intermediate risk	High risk
Dental Eye Gynecologic Breast Minor genitourinary	Head and neck Transplant Major genitourinary Intraperitoneal Intrathoracic	Open heart Vascular

Box 1-2 Assessment of functional capacity

Metabolic equivalents of task (METs) are a physiologic measurement that expresses the energy associated with physical activities.

1 MET

- Can you take care of yourself?
- Can you walk indoors?
- Can you feed yourself?
- Can you dress yourself?
- Can you walk 1 to 2 blocks?

4 METs

- Can you climb a flight of stairs?
- Can you do heavy housework?
- Can you participate in moderate recreational activities?

> 10 METs

- Can you participate in strenuous sports?

Box 1-3 Cardiac risk factors

- History of angina
- History of myocardial infarction
- History of heart failure
- History of stroke
- Diabetes mellitus
- Renal failure

Respiratory Disease

Normal Lung Volumes (Figs 1-2 and 1-3)

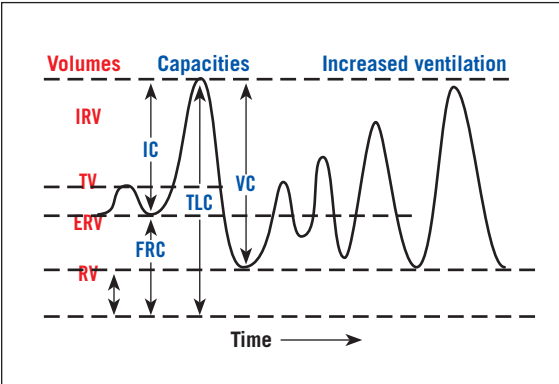


Fig 1-2 Lung volumes and capacities. IRV, inspirational reserve capacity; TV, tidal volume; ERV, expiratory reserve volume; RV, residual volume; IC, inspirational capacity; FRC, functional residual capacity; TLC, total lung capacity; VC, vital capacity.

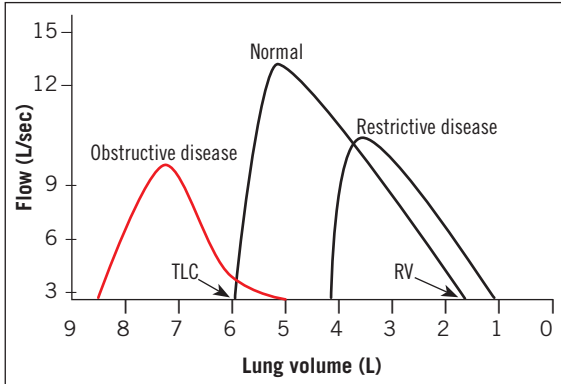


Fig 1-3 Flow volume curves. (Reprinted with permission from Levitzky MG. Pulmonary Physiology, 7 ed. New York: McGraw-Hill, 2007.)

Common abbreviations

- Residual volume (RV): Air left after maximal expiration
- Tidal volume (TV): Entering air during normal inspiration
- Expiratory reserve volume (ERV): Air that can still be expired after normal expiration
- Functional residual capacity (FRC): $RV + ERV$

Abbreviations associated with pulmonary function tests (PFTs)

- Forced expiratory volume in 1 second (FEV1): Air that can be expired in 1 second
- Forced vital capacity (FVC): Maximum volume of air that can be forcefully exhaled
- Total lung capacity (TLC): $FVC + RV$

Common types of pulmonary disease

Obstructive	Restrictive	Extraparenchymal restrictive
<ul style="list-style-type: none"> • Asthma • Cystic fibrosis • Chronic obstructive pulmonary disease (COPD) 	<ul style="list-style-type: none"> • Sarcoidosis • Interstitial lung disease • Collagen disorder 	<ul style="list-style-type: none"> • Obesity • Scoliosis • Myasthenia gravis • Diaphragmatic weakness • Cervical spine injury

Obstructive versus restrictive lung disease

	Obstructive	Restrictive
FEV1	Decreased	Decreased
FVC	Normal	Decreased
FEV1/FVC	Decreased	Normal/increased
Lung volume	Increased	Decreased
Flow rates	Decreased	Decreased

Chronic Respiratory Diseases

Asthma

- **Definition:** Chronic obstructive reversible disorder of airway hyper-reactivity causing dyspnea, cough, wheezing, and chest tightness
- **Diagnosis:** Diagnosed by showing reversible obstructive lung disease with normal diffusing capacity
- Exam will show expiratory wheezing during acute exacerbations, with a prolonged expiratory phase
- Severe attacks will have pulsus paradoxus, accessory muscle use, and silent chest

	Definition	Treatment
Mild intermittent	< 2 days/week with PEF > 80%	Bronchodilator as needed
Mild persistent	> 2 days/week but < 1 time/day with PEF > 80%	Low-dose inhaled steroids
Moderate persistent	Daily symptoms with PEF between 60% and 80%	Inhaled steroids and long-acting beta 2 agonist
Severe persistent	Continuous symptoms with PEF < 60%	Add oral steroids

PEF, peak expiratory flow.

Medications for treatment of asthma

	Mechanism of action	Example
Beta 2 agonist	Beta 2 agonism causes an increase in cAMP formation leading to relaxation of bronchial muscle	Albuterol, salmeterol
Corticosteroids	Suppresses inflammatory response and decreases mucosal edema	Fluticasone, hydrocortisone, prednisolone
Leukotriene modifier	Leukotriene receptor antagonist decreases bronchoconstriction	Montelukast

(Medications for treatment of asthma cont)

	Mechanism of action	Example
5-Lipoxygenase	Inhibits leukotriene formation	Zileuton
Anticholinergic	Blocks cholinergic constriction causing bronchodilation	Ipratropium bromide
cAMP, cyclic adenosine monophosphate.		

Chronic Obstructive Pulmonary Disease (COPD)

- **Definition:** Nonreversible chronic airway restriction
- **Symptoms:** Worsening dyspnea, increasing cough and change in sputum, hyperinflation, prolonged expiration, wheezing
- **Chronic bronchitis:** Chronic productive cough for 3 months in 2 consecutive years; “blue bloater”
- **Emphysema:** Enlargement of airways and wall destruction distal to bronchioles; “pink puffer;” pursed-lip breathing
- **Diagnosis:**
 - PFTs to evaluate FEV1, FEV1/FVC, and postbronchodilator values
 - Arterial blood gas (ABG) analysis will show hypercarbia, hypoxemia
 - Evaluate for alpha 1 antitrypsin deficiency in emphysema patients
 - Chest radiograph

Classification of COPD

Stage	FEV1	Treatment
1	> 80	Short-acting bronchodilator (albuterol)
2	50–79	Long-acting B2 agonist (salmeterol) and anticholinergic bronchodilator (ipratropium)
3	30–49	Inhaled steroid
4	< 30	Oxygen, pulmonary rehabilitation, consider transplant in worst cases

Acute Pulmonary Diseases

Acute respiratory distress syndrome (ARDS)

- **Definition:** Acute, hypoxemic respiratory failure associated with bilateral lung infiltrates
- **Etiology:** Pneumonia, aspiration, trauma, acute pancreatitis, inhalational injury, reperfusion injury
- **Symptoms:** Rapid onset of dyspnea, tachypnea, diffuse lung crackles
- **Diagnosis:** Bilateral infiltrates on chest radiograph, ratio of PaO₂ to FiO₂ < 200
- **Treatment**
 - Treat underlying cause
 - Use mechanical ventilation with low tidal volumes of 6 cc/kg
 - Positive end-expiratory pressure (PEEP)
 - Conservative fluid management

Pulmonary embolus (PE)

- **Risk factors:** Prior PE, pregnancy, malignancy, obesity, immobility, stroke, tobacco use, recent surgery, trauma
- **Symptoms:** Dyspnea, hemoptysis, fever, cough, tachypnea, tachycardia
- **Diagnosis**
 - Modified Wells criteria (see table below)
 - D-dimer test: Only helpful to exclude PE in low-risk patients (Wells score ≤ 4)
 - Computed tomography angiography (CTA): Multidetector-row CTA (MDCTA) is standard pulmonary angiography when CTA is not available
 - ECG: New right heart strain; nonspecific anterior T wave inversions; sinus tachycardia; large S wave in lead I, a large Q wave in lead III, and an inverted T wave in lead III (S1Q3T3)
 - ABG analysis: Respiratory alkalosis with increased alveolar arterial gradient
 - V/Q scan: Ventilation without perfusion suggests PE
- **Treatment**
 - Heparin as bridge to coumadin to maintain INR of 2 to 3 for at least 3 to 6 months
 - Inferior vena cava filter if anticoagulation is contraindicated
 - Direct thrombin inhibitors for patients with heparin-induced thrombocytopenia (HIT)
 - Thrombolysis for massive PE
 - Thrombectomy

Modified Wells criteria

To determine likelihood of PE.

- ≤ 4 = Unlikely PE
- > 4 = Likely PE

Criteria	Points
Clinical signs and symptoms of DVT	3
PE is primary diagnosis	3
Heart rate > 100 bpm	1.5
Immobilized for at least 3 days or surgery in previous 4 weeks	1.5
Previous objectively diagnosed PE or DVT	1.5
Malignancy with treatment within 6 months or palliation	1
Hemoptysis	1
DVT, deep vein thrombosis.	

Contraindications to anticoagulation

Relative	Absolute
<ul style="list-style-type: none"> • Thrombocytopenia • Prior hemorrhagic stroke • Recent internal bleeding 	<ul style="list-style-type: none"> • Active internal bleeding • Aortic dissection • Active hemorrhagic stroke

Renal Disease

Acute Renal Failure

- Increase in serum creatinine ≥ 0.3 mg/dL over baseline
- Urine output less than 0.5 cc/kg/hour for more than 6 to 12 hours

Prerenal

- **Etiology:** Volume depletion, severe liver disease, severe CHF
- **Diagnosis:** Fractional excretion of sodium (FENa) $< 1\%$; ratio of blood urea nitrogen (BUN) to creatinine, 10–15:1; high urinary osmolarity
- **Treatment:** Fluids (rapid improvement with fluids)

Renal

- **Etiology:** Tubular injury, acute tubular necrosis, interstitial disease, glomerular disorder
- **Diagnosis:** FENa $> 1\%$; BUN-to-creatinine ratio, 10–15:1; muddy brown casts
- **Treatment:** Remove underlying agent, treat underlying cause

Postrenal

- **Etiology:** Urinary tract obstruction
- **Diagnosis:** FENa $< 1\%$, oliguria/anuria
- **Treatment:** Remove obstruction

Need for emergent dialysis

A	Acidosis
E	Electrolyte abnormality
I	Ingestion
O	Overload
U	Uremia

Chronic Renal Failure (CRF)

- Permanent loss of renal function for at least 3 months
- **Etiology:** Hypertension, diabetes, renal artery stenosis, polycystic kidney disease
- **Diagnosis:** Glomerular filtration rate (GFR) < 15 mL/minute, albuminuria > 30 mg/day
- **Treatment:** Management of hypertension with ACE inhibitors and angiotensin receptor blockers, low density lipoproteins < 100 mg/dL
- Predictor of disease progression: Proteinuria

Severity of chronic renal disease based on GFR

GFR stage	GFR (mL/min/1.73 m ²)	Kidney function
G1	> 90	Normal
G2	60–89	Mildly decreased
G3a	45–59	Mild to moderately decreased
G3b	30–44	Moderately to severely decreased
G4	15–29	Severely decreased
G5	< 15	Kidney failure
G5D	< 15	Kidney failure treated with dialysis

Severity of chronic renal disease based on albuminuria

Albuminuria stage	Albumin excretion rate (mg/day)	Albumin excretion
A1	< 30	Normal to increased
A2	30–300	Moderately increased
A3	> 300	Severely increased

Complications of CRF

Treatment	
Anemia	Erythropoietin injections, iron
Renal osteodystrophy	Phosphate binder
Hyperkalemia	Dietary restriction, diuretic
Acidosis	Sodium bicarbonate
Pericarditis	Dialysis
Dialysis infections	Antibiotics, catheter removal

Urinalysis interpretation

Marker	Normal	Abnormal	Cause of abnormality
Turbidity	Clear	Turbid	Crystals or cells present
Color	Yellow	Brown to red	Myoglobin/hemoglobin
Leukocyte esterase (LE)	Negative	Present	Lysed neutrophils and macrophages elicit LE (WBC marker)
Nitrite	Negative	Present	Underlying bacteriuria
Protein	< 30 mg/dL	> 30 mg/dL	Renal failure
Specific gravity	1.008–1.009	Outside of normal range	Renal failure, dehydration, renal artery stenosis
Glucose	Negative	Present	Renal failure or elevated serum glucose
WBC, white blood cell.			

Nephrotic Disease

- **Symptoms:** Peripheral edema, hypoalbuminemia, hyperlipidemia, increased proteinuria
- **Diagnosis:** Urinalysis shows oval fat bodies, proteinuria, and 24-hour urine protein > 3.5 g/day

Primary nephrotic syndrome

Direct damage to glomeruli causing massive proteinuria.

Primary disease	Pathology	Treatment
Membranous nephropathy	Thickening of capillary loops with subepithelial deposits	ACE inhibitor, steroid
Focal segmental glomerulosclerosis	Glomerulosclerosis	Steroids, cyclosporine
Goodpasture syndrome	Linear IgG deposition along glomerular basement membrane	Steroids, cyclophosphamide, plasmapheresis
Minimal change disease	Epithelial foot process loss	Steroids
IgG, immunoglobulin G.		

Secondary nephrotic syndrome

Damage of glomeruli secondary to systemic disease.

	Pathology	Treatment
Diabetes mellitus	Kimmelstiel-Wilson lesion	Glucose, lipid, blood pressure control, ACE inhibitor, angiotensin receptor blocker
Multiple myeloma	Light chain involvement with positive Congo red stain	Treatment of systemic disease
Amyloidosis	Amyloid deposition with positive Congo red stain	Treatment of systemic disease

Nephritic Disease

Inflammatory disorder in the glomeruli.

- Glomerulonephritis: RBCs in urine with or without cellular casts and varying degrees of proteinuria
- **Symptoms:** Hypertension, edema, oliguria, hematuria
- **Diagnosis:** Red blood cell (RBC) casts in urine, renal biopsy
- Types of glomerulonephritis
 - Immune complex: Decreased complement levels
 - Pauci immune: Normal complement levels

Immune complex glomerulonephritis

	Pathology	Treatment
Subacute bacterial endocarditis	Crescent glomerulonephritis	Antibiotics
Post-streptococcal	Subepithelial humps	Resolves after treatment of streptococcal infection
Membranoproliferative glomerulonephritis	Subendothelial deposits	Treat cryoglobulinemia

Pauci immune glomerulonephritis

	Pathology	Treatment
IgA nephropathy	IgA deposits in mesangium	ACE inhibitor/angiotensin receptor blocker, steroids
Wegener granulomatosis	Necrotizing crescent disease	Steroids, cyclophosphamide
Churg-Strauss syndrome	Necrotizing crescent disease	Steroids, cyclophosphamide
IgA, immunoglobulin A.		

Nephrotic Versus Nephritic Disease

	Nephrotic	Nephritic
Protein	Very large amount	Small amount
Urinalysis	No casts but will find lipid-laden macrophages and free lipid	Abundant RBCs and RBC casts; no lipids seen
BP	Mildly elevated or normal	Severely elevated
GFR	Normal	Elevated

BP, blood pressure.

Acid-Base Disorders

Arterial Blood Gas (ABG) Versus Venous Blood Gas (VBG)

- ABG: The gold standard to evaluate acid-base disorders
 - Invasive procedure
 - Serial examinations necessary
 - Risk of hematoma and nerve injury
- VBG
 - Easier to obtain and less injury to patients
 - Data (pH, bicarbonate [HCO_3^-], lactate, and base excess) are similar to those found in ABG
 - Partial pressure of carbon dioxide (PaCO_2) is also well correlated except in patients with severe shock or when $\text{PaCO}_2 > 45$ mm Hg

Quick Guide to ABG Interpretation

Step one: Identify primary disorder

- Evaluate pH and PaCO_2 :
 - If change in same direction → metabolic disorder
 - If change in different direction → respiratory disorder

Primary disorder	Primary change	Compensatory change
Metabolic acidosis	Decreased HCO_3^-	Decreased PaCO_2
Metabolic alkalosis	Increased HCO_3^-	Increased PaCO_2
Respiratory acidosis	Increased PaCO_2	Increased HCO_3^-
Respiratory alkalosis	Decreased PaCO_2	Decreased HCO_3^-

Step two: Check for compensation if disorder has a primary origin

- Metabolic disorder: Calculate the expected PaCO_2
- Respiratory disorder: Calculate the expected pH
- If the actual value is different from the calculated value (pH or PaCO_2), expect an additional acid-base disorder

Step three: Calculate the anion gap if metabolic acidosis or mixed disorder is detected

- Anion gap (AG): $\text{Na} - (\text{Cl} + \text{HCO}_3) \leq 12$
- If $\text{AG} < 12$, acidosis is due to loss of bicarbonate (ie, diarrhea)
- If $\text{AG} > 12$, acidosis is due to increase of nonvolatile acids (ie, lactic acidosis)
- AG can be influenced by an abnormal albumin level

Acid-Base Disorders

Metabolic acidosis	<p>Decreased blood pH with decreased bicarbonate</p> <p>Etiology: Two types</p> <ul style="list-style-type: none"> • $\text{AG} > 12$: "MUDPILES" <ul style="list-style-type: none"> – Methanol ingestion – Uremia – Diabetic ketoacidosis – Paraldehyde Ingestion – Isoniazid ingestion – Lactic acidosis – Ethylene glycol ingestion – Salicylate ingestion • Non-AG <ul style="list-style-type: none"> – Gastrointestinal losses: Diarrhea, small bowel fistula, pancreatic fistula – Renal loss: Renal tubular acidosis <p>Signs/Symptoms</p> <ul style="list-style-type: none"> • Hyperventilation (compensatory mechanism) • Decreased tissue perfusion • Decreased cardiac output • Altered mental status • Arrhythmias • Hyperkalemia <p>Treatment</p> <ul style="list-style-type: none"> • Treat underlying cause <ul style="list-style-type: none"> – Most of the time acidosis is not harmful – Cause of death in these patients due to underlying condition rather than acidemia • Sodium bicarbonate <ul style="list-style-type: none"> – Has shown to be ineffective therapy in management of acidosis – Only use in patients who are deteriorating rapidly
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(Acid-Base Disorders cont)

Metabolic alkalosis	<p>Increased blood pH with increased bicarbonate</p> <p>Etiology</p> <ul style="list-style-type: none"> • Extracellular fluid expansion <ul style="list-style-type: none"> – Adrenal disorders causing increased mineralocorticoid secretion; increased reabsorption of bicarbonate and sodium and secretion of chloride • Extracellular fluid contraction <ul style="list-style-type: none"> – Vomiting, nasogastric suction causing hydrochloric acid and bicarbonate loss – Excessive use of diuretics <p>Signs/Symptoms</p> <ul style="list-style-type: none"> • Hypokalemia • Elevated bicarbonate • Elevated pH • Hypoventilation • Arrhythmias • Decrease in cerebral blood flow <p>Treatment</p> <ul style="list-style-type: none"> • Treat underlying cause • Volume-depleted patient requires normal saline with potassium replacement • In the volume-overloaded patient, consider spironolactone
Respiratory acidosis	<p>Alveolar hypoventilation: Decreased blood pH with arterial $\text{PaCO}_2 > 40$</p> <ul style="list-style-type: none"> • Acute: No renal compensation • Chronic: Renal compensation with increase in plasma bicarbonate <p>Etiology</p> <ul style="list-style-type: none"> • Chronic obstructive pulmonary disease • Brainstem injury • Respiratory muscle fatigue • Drug overdose causing hypoventilation <p>Signs/Symptoms</p> <ul style="list-style-type: none"> • Confusion • Headaches • Fatigue • Central nervous system (CNS) depression <p>Treatment</p> <ul style="list-style-type: none"> • Supplemental oxygen • Treat underlying disorder • Consider mechanical ventilation with severe acidosis, deteriorating mental status, and impending respiratory failure

(Acid-Base Disorders cont)

Respiratory alkalosis	<p>Alveolar hyperventilation: Increased blood pH with decrease in PaCO₂</p> <ul style="list-style-type: none"> • Acute renal compensation: For every 10 mm Hg decrease in PaCO₂, bicarbonate will decrease by 2 • Chronic renal compensation: For every 10 mm Hg decrease in PaCO₂, bicarbonate will decrease by 5 <p>Etiology</p> <ul style="list-style-type: none"> • Anxiety • Sepsis • Pregnancy • Liver disease • Pulmonary embolism • Asthma <p>Signs/Symptoms</p> <ul style="list-style-type: none"> • Decreased cerebral blood flow • Lightheadedness • Anxiety • Perioral numbness • Arrhythmias <p>Treatment</p> <ul style="list-style-type: none"> • Treat underlying disorder • Inhale CO₂ (breathing into a paper bag)
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Sodium Disorders

- Normal sodium concentration in the body is 135 to 145 mEq/L
- To determine the cause of sodium disorder, measure
 - Plasma osmolality (290 mOsm/kg H₂O)
 - $(2 \times \text{Plasma Na}^+) + \text{Glucose}/18$
 - Extracellular volume
 - Clinical examination (eg, peripheral edema, orthostatic hypotension, and skin turgor)
 - Not the most reliable method but is readily available
 - Invasive monitoring (cardiac filling pressures and cardiac output)

Hyponatremia	<p>Symptoms</p> <ul style="list-style-type: none"> • Lethargy, seizures, nausea/vomiting, confusion <p>Diagnosis/Etiology</p> <ul style="list-style-type: none"> • Measure serum osmolality <ul style="list-style-type: none"> – Normal: Isotonic hyponatremia (pseudohyponatremia) <ul style="list-style-type: none"> ◦ Hyperlipidemia ◦ Hyperproteinemia – High: Hypertonic hyponatremia; hyperglycemia – Low: Hypotonic hyponatremia; measure volume status <ul style="list-style-type: none"> ◦ Hypovolemic <ul style="list-style-type: none"> – Extrarenal salt loss: Urine sodium low ~10 mEq/L; diarrhea/vomiting – Renal salt loss: Urine sodium high ~20 mEq/L; acute tubular necrosis or excessive diuretic use
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(Sodium Disorders cont)

Hyponatremia (cont)	<ul style="list-style-type: none"> ◦ Euvolemic <ul style="list-style-type: none"> – Syndrome of inappropriate antidiuretic hormone secretion (SIADH): < 20 mEq/L urine sodium, < 100 mOsm/kg H_2O urine osmolality – Psychogenic polydipsia: > 10 mEq/L urine sodium, > 100 mOsm/kg H_2O urine osmolality – Hypothyroid disease ◦ Hypervolemic <ul style="list-style-type: none"> – CHF/liver disease: > 20 mEq/L urine sodium – Renal failure: < 20 mEq/L urine sodium <p>Treatment</p> <ul style="list-style-type: none"> • Based on volume status and neurologic symptoms • Rapid normalization of sodium level can lead to demyelinating encephalopathy <ul style="list-style-type: none"> – Plasma rise should not exceed 0.5 mEq/L per hour • Amount of replacement can be guided by calculation of sodium deficit <ul style="list-style-type: none"> – Sodium deficit = normal total body water \times (130 – current plasma sodium) • Hypotonic – Hypovolemia: Hypertonic saline (3% NaCl) in symptomatic patients; isotonic saline in asymptomatic patients <ul style="list-style-type: none"> – Euvolemia: Combined furosemide diuresis and infusion of hypotonic saline in symptomatic patients; isotonic saline in asymptomatic patients – Hypervolemia: Diuretics with addition of hypertonic saline only in symptomatic patients
Hypernatremia	<p>Symptoms</p> <ul style="list-style-type: none"> • Lethargy, weakness, irritability, seizure, polyuria <p>Diagnosis/Etiology</p> <ul style="list-style-type: none"> • Measure volume status – Hypovolemic: More water loss than sodium loss <ul style="list-style-type: none"> ◦ Renal loss <ul style="list-style-type: none"> – Renal failure – Diuretics – Non-ketotic hyperglycemia (NKH): plasma glucose usually $> 1,000$ mg/dL ◦ Nonrenal loss – Diarrhea – Respiratory loss – Euvolemic: Loss of water only <ul style="list-style-type: none"> ◦ Diabetes insipidus: Central versus neurogenic – Hypervolemic: More sodium gain than water gain <ul style="list-style-type: none"> ◦ Excess of mineralocorticoids ◦ Cushing syndrome <p>Treatment</p> <ul style="list-style-type: none"> • Calculate the free water deficit and replace with isotonic fluid • Treat underlying causes – Central diabetes insipidus: Treat with vasopressin 2 to 5 units subcutaneously every 4 to 6 hours

Potassium Disorders

- Normal potassium level is between 3.5 mEq/L and 5 mEq/L
- Work-up should include:
 - Urine potassium and chloride level
 - Serum magnesium level
 - ABG as needed

Hypokalemia	<p>Hypokalemia is better tolerated than hyperkalemia</p> <p>Etiology</p> <ul style="list-style-type: none"> • Nonrenal: Urine potassium < 30 mEq/L; diarrhea • Renal loss: Urine potassium > 30 mEq/L <ul style="list-style-type: none"> – High urine chloride (> 25 mEq/L) <ul style="list-style-type: none"> ◦ Magnesium depletion ◦ Diuretic – Low urine chloride (< 25 mEq/L) <ul style="list-style-type: none"> ◦ Nasogastric suctioning ◦ Alkalosis <p>Symptoms</p> <ul style="list-style-type: none"> • Mild hypokalemia (2.5 to 3.5); can be asymptomatic • Severe hypokalemia (< 2.5 mEq/L); diffuse muscle weakness • Abnormal ECG; prominent U waves, flattening and inversion of T waves, and QT prolongation • Only occurs in 50% of cases <p>Treatment</p> <ul style="list-style-type: none"> • Treat underlying cause • Magnesium replacement • Potassium (KCl) replacement should be done gradually <ul style="list-style-type: none"> – Oral replacement in mild cases – Intravenous (IV) replacement in cases with arrhythmia; increase no greater than 20 mEq/L
Hyperkalemia	<p>Poorly tolerated, especially when level is above 5.5 mEq/L; a patient with chronic renal disease may normally have an elevated potassium level</p> <p>Etiology</p> <ul style="list-style-type: none"> • Nonrenal (transcellular shift) <ul style="list-style-type: none"> – Acidosis – Rhabdomyolysis • Impaired renal excretion <ul style="list-style-type: none"> – Adrenal insufficiency – Drug (eg, potassium-sparing diuretics) – Renal insufficiency

(Potassium Disorders cont)

Hyperkalemia (cont)	Symptoms <ul style="list-style-type: none">• ECG changes – Begins to change when potassium reaches 6 mEq/L – Stages<ul style="list-style-type: none">◦ 1st stage: Peaked T waves (V2 and V3)◦ 2nd stage: Flattened P waves and PR interval lengthening◦ 3rd stage: Disappearance of P waves and QRS prolongation◦ Final: Ventricular asystole• Respiratory failure• Nausea/vomiting• Muscle weakness Treatment <ul style="list-style-type: none">• ECG changes: IV calcium gluconate to decrease cardiac excitability• Shift potassium from extracellular to intracellular with insulin and dextrose• Diuretics, exchange resins (kayexalate), dialysis to remove potassium
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Gastrointestinal Disease

Irritable Bowel Syndrome

	Crohn disease	Ulcerative colitis
Definition	Chronic disease with patchy transmural inflammation	Chronic disease with diffuse and continuous mucosal inflammation
Symptoms	Nonbloody diarrhea, low-grade fever, pain, malaise, weight loss	Bloody diarrhea, fecal urgency, fever, uveitis, erythema nodosum, ankylosing spondylitis
Location	Anywhere in the gastrointestinal tract with propensity for the ileum	Colon to the rectum
Diagnosis	Colonoscopy with biopsy	Colonoscopy with biopsy, stool studies, abdominal radiograph showing lead pipe appearance of colon with loss of haustrations
Malignancy potential	Questionable increased malignancy risk	Increased malignancy risk
Treatment	Steroid, immunomodulatory drugs, 5-aminosalicylic acid	Mesalamine, steroid, surgery

Hepatitis

	Hepatitis A	Hepatitis B	Hepatitis C
Diagnosis	Antihepatitis A virus (IgM versus IgG)	Active infection: Surface antigen Prior infection: Antihepatitis B core	Antihepatitis C or check polymerase chain reaction for virus
Transmission	Fecal or oral transmission	Blood to blood transmission	Blood to blood transmission
Treatment	Supportive care	Follow liver function tests and treat when necessary; options include nucleoside analogs, pegylated interferon, and liver transplantation at end stage	Interferon, ribavirin, transplantation at end stage
IgM, immunoglobulin M; IgG, immunoglobulin G.			

Hepatitis B serology

	Hepatitis B surface antigen	Antihepatitis B surface antigen	Antihepatitis B core antigen	Hepatitis B core antigen
Acute hepatitis B	Positive	Negative	Positive (IgM)	Positive
Chronic hepatitis B	Positive	Negative	Positive (IgG)	Positive if virus is actively replicating
Vaccination	Negative	Positive	Negative	Negative
IgM, immunoglobulin M; IgG, immunoglobulin G.				

Gastroesophageal Reflux Disease

- **Etiology:** Lower esophageal sphincter relaxation
- **Symptoms:** Retrosternal burning, regurgitation, excessive salivation, bitter test, throat fullness, halitosis
- **Diagnosis:** Treat empirically; if no success, upper endoscopy with biopsy, esophageal pH monitoring
- **Treatment:** Elevate head of bed, stop tobacco and alcohol use, dietary modification, antacids, histamine blockers, proton pump inhibitors
- **Complications:** Barrett esophagus, adenocarcinoma, upper gastrointestinal bleeding, cough, asthma

End Stage Liver Disease (ESLD)

- **Etiology:** Chronic hepatocellular injury leads to fibrosis of liver
- **Symptoms:** Fatigue, anorexia, impotence, melena, spider nevi, gynecomastia, jaundice, testicular atrophy, coarse hand tremor, caput medusae, spider telangiectasia, Dupuytren contractures
- **Diagnosis:** Liver function tests, liver biopsy; monitor disease with Child-Turcotte-Pugh score or model for end stage liver disease (MELD) score

- **Treatment:** Avoid alcohol and medications metabolized by the liver, treat underlying disease process, screen for hepatocellular carcinoma, monitor for complications
- **Complications:** Esophageal varices, ascites, increase in bleeding risk, portal hypertension, hepatic encephalopathy

Child-Turcotte-Pugh score

- 5 to 6 points: Class A, 90% 3-year survival rate
- 7 to 9 points: Class B, 50% to 60% 3-year survival rate
- > 9 points: Class C, 30% 3-year survival rate

	1 Point	2 Points	3 Points
Ascites	Absent	Present but not tense	Tense abdomen
Encephalopathy	Absent	Grades 1 or 2	Grades 3 or 4
Albumin	> 3.5 g/dL	2.8–3.5 g/dL	< 2.8 g/dL
Prothrombin time (over normal)	1–3 min	4–6 min	> 6 min
Bilirubin	< 2 mg/dL	2–3 mg/dL	> 3 mg/dL

Hematologic Disease

Anemia

Hypoproliferative (low reticular cell count)

	Microcytic (MCV < 80 fL)	Normocytic (MCV 80–100 fL)	Macrocytic (MCV > 100 fL)
Example	Iron deficiency	Renal insufficiency	B12/folate deficiency
Diagnosis	Microcytic, hypochromic with anisocytosis, low ferritin	Increased creatinine associated with anemia	Low B12/folate, macrocytic, hypersegmented neutrophils, decreased intrinsic factor; Schilling test to establish cause of B12 deficiency
Symptoms	Pica, ice chip cravings, glossitis, koilonychias, cheilosis	Anemia symptoms amidst underlying renal disorder	Glossitis, atrophic gastritis, neurologic symptoms in severe cases
Treatment	Iron therapy	<ul style="list-style-type: none">• Erythropoietin administration• Iron dosing	B12/folate dosing

MCV, mean cell volume.

Hyperproliferative (high reticular cell count)

	Hemolytic		Nonhemolytic
	Intravascular	Extravascular	Bleeding
Site of RBC destruction	Bloodstream/liver	Spleen	Secondary to blood loss
Diagnosis on smear	Schistocytes	Spherocytes	N/A
Example	<ul style="list-style-type: none"> Cold Ab immune hemolysis G6PD Deficiency 	<ul style="list-style-type: none"> Warm Ab immune hemolysis Hypersplenism 	Surgical/traumatic bleeding
Ab, antibody; G6PD, glucose-6-phosphate dehydrogenase.			

Sickle Cell Disease

Homozygous defect in gene for beta-globulin that produces hemoglobin S.

- **Triggers:** Dehydration, acidosis, hypoxia
- **Diagnosis:** Target cells, sickle cells, Howell Jolly bodies, hemoglobin S on smear
- **Symptoms:** Acute chest pain, stroke, autosplenectomy
- **Treatment:** Folate, hydroxyurea, aggressive hydration, analgesia, oxygen, transfuse for major surgery (9 to 10 g hemoglobin)

Acute complications	Chronic complications
<ul style="list-style-type: none"> Stroke Splenic infarct Osteomyelitis 	<ul style="list-style-type: none"> Retinopathy Avascular necrosis of the hip Chronic renal failure

Bleeding Disorders

	Factor 8 deficiency (hemophilia A)	Factor 9 deficiency (hemophilia B)	vWF deficiency (von Willebrand disease)	Immune thrombocytopenic purpura
Symptom	Spontaneous bleeding	Spontaneous bleeding, prolonged PTT	Petechiae, mucosal bleeding, epistaxis, factor 8 activity with vWF deficiency	Normal spleen with petechiae and mucosal bleeding due to immune destruction of platelets
Treatment	Treat with desmopressin or factor replacement	Treat with factor replacement	Treat with desmopressin, aminocaproic acid, or factor 8 replacement*	Treat with platelet transfusion if platelets drop below 25,000–30,000 mL
vWF, von Willebrand factor; PTT, partial thromboplastin time. *Avoid aspirin and nonsteroidal anti-inflammatory drugs—can prolong bleeding.				

Disseminated Intravascular Coagulation (DIC)

Consumptive coagulopathy associated with serious illness.

- **Symptoms:** Thrombocytopenia, excessive bleeding or clotting
- **Diagnosis:** Decreased fibrinogen, platelets; increased prothrombin time (PT)/ partial thromboplastin time (PTT), d-dimer test; schistocytes present
- **Treatment:** Treat underlying cause; platelets and cryoprecipitate for bleeding, low-dose heparin for clotting

Hypercoagulable State

- **Risk factors:** Prior embolus, pregnancy, surgery, tobacco use, prolonged immobilization, hospitalization, malignancy
- **Diagnosis:** History and physical examination, complete blood count, PTT
- **Treatment:** Postoperative patients should be treated for 3 months at an INR of 2 to 3, all others for 3 to 6 months
- **Exceptions**
 - Active cancer: Treat for duration of disease
 - Mechanical heart valves: INR goal is 3 to 4

Specific thrombophilic disorders

Disease	Thrombosis
Factor V Leiden	Venous
Protein C/S deficiency	Arterial and venous
Heparin-induced thrombocytopenia (HIT)	Arterial and venous

Anticoagulation medications

	Laboratory check	Reversibility
Warfarin	INR	Fresh frozen plasma, vitamin K
Heparin	PTT and platelet count (monitor for HIT)	Protamine
Low-molecular-weight heparin	Antifactor Xa	No
Fondaparinux (factor Xa inhibitors)	No monitoring	No
Dabigatran (direct thrombin inhibitors)	PTT	No

Endocrine Disease

Diabetes Mellitus (DM)

Diagnosis

- Random glucose > 200 mg/dL
- Fasting glucose > 126 mg/dL
- Two-hour glucose > 200 mg/dL (75 gm)
- Hemoglobin A1c (HbA1c) > 6.5

	Type 1 DM	Type 2 DM
Symptoms	Polyuria, polydipsia, polyphagia	Mild or none
Stature	Skinny	Obese
Etiology	Autoimmune islet cell destruction	Insulin resistance associated with obesity
Treatment	Insulin therapy, glycemic control, lifestyle management	Oral hypoglycemic, glycemic control, lifestyle management
Complication	Diabetic ketoacidosis	Hyperosmolar nonketotic coma
Chronic complications	Retinopathy, neuropathy, nephropathy, infections, myocardial infarction, cardiovascular disease, stroke	

Insulin types

	Onset	Peak	Duration
Ultra short acting (lispro)	5–15 min	60–90 min	3–4 hr
Short acting (regular)	15–30 min	1–3 hr	5–7 hr
Intermediate acting (NPH)	2–4 hr	8–10 hr	18–24 hr
Long acting (glargine)	3–4 hr	N/A	24 hr
NPH, neutral protamine hagedorn.			

Oral hypoglycemics

	Mechanism	Notes
Biguanide (metformin)	Decreases insulin resistance and glucose production	Can cause lactic acidosis, gastrointestinal upset
Sulfonylurea (glyburide)	Stimulates insulin release	Can cause hypoglycemia
Meglitinide (repaglinide)	Stimulates pancreas to release insulin	Can cause weight gain
Thiazolidinedione (pioglitazone)	Decreases insulin resistance peripherally	Causes retention of fluid

Goals of treatment

- BP < 130/85 mm Hg
- Low-density lipoprotein < 100 mg/dL, total glycerides < 150 mg/dL, high-density lipoprotein > 40 mg/dL
- Smoking cessation
- Glycemic control for HbA1c < 7

Monitoring glycemic control in DM

- HbA1c >10 is poor control
- HbA1c between 8.5 and 10 is fair control
- HbA1c between 7 and 8.5 is good control
- Fasting glucose < 130 mg/dL
- Peak postprandial glucose < 180 mg/dL

Diabetic Ketoacidosis

An insulin deficiency and glucagon excess that causes severe hyperglycemia and ketogenesis. Severe hyperglycemia causes an osmotic diuresis leading to dehydration and volume depletion.

- **Symptoms:** Abdominal pain, nausea, vomiting, Kussmaul respirations, ketone breath, anion gap metabolic acidosis, marked dehydration, tachycardia, polydipsia, polyuria, weakness, altered consciousness
- **Diagnosis:** Serum glucose > 250 mg/dL, metabolic acidosis (pH > 7.3 and serum bicarbonate < 15 mEq/L), increased anion gap, ketonuria, ketonemia; check chemistry panel for hyperkalemia and hyponatremia
- **Treatment:**
 - IV insulin dose at 0.1 units/kg, then start drip at 0.1 units/kg/hour (check potassium prior to starting insulin); drip should run with normal saline replacement
 - Once anion gap has closed and acidosis is resolved, start to decrease the insulin and switch to subcutaneous insulin
 - Add dextrose to IV fluids when glucose is below 250 mg/dL
 - Manage sodium, potassium, and magnesium levels very closely

Thyroid Disorders

	Hypothyroid	Hyperthyroid
Diagnosis	Elevated TSH and decreased T4	Decreased TSH and increased T4
Symptoms	Fatigue, weight gain, cold intolerance, depression	Palpitations, heat intolerance, sweating, anxiety
Examples	Hashimoto thyroiditis, subacute thyroiditis, iodine deficiency	Graves disease, toxic nodule, goiter
Treatment	Synthroid	Ablation surgery, propylthiouracil, methimazole
Complications	Myxedema coma with hypercapnia, hypoventilation, hypothermia	Atrial fibrillation, thyroid storm
TSH, thyroid-stimulating hormone; T4, thyroxine.		

Adrenal Disorders (Fig 1-4)

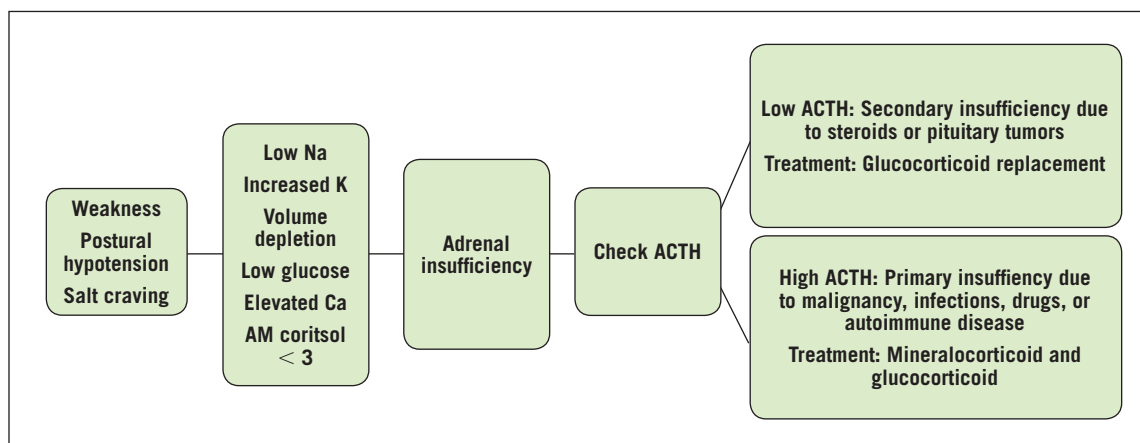


Fig 1-4 Diagnostic algorithm for adrenal disorders. Na, sodium; K, potassium; Ca, calcium; AM, morning; ACTH, adrenocorticotrophic hormone.

Complications: Adrenal crisis—shock, nausea, vomiting, confusion, fever; can be fatal

Addison disease (primary adrenal insufficiency)

- **Etiology:** Autoimmune adrenalitis, malignancy, infection
- **Symptoms:** Hyperpigmentation of the oral mucosa, dehydration, hypotension, fatigue, anorexia, nausea, vomiting, diarrhea, abdominal pain, salt craving, hyponatremia, hyperkalemia
- **Diagnosis:** Check chemistry panel for electrolyte abnormalities, low cortisol, high adrenocorticotrophic hormone (ACTH)
- **Treatment:** Mineralocorticoid and glucocorticoid replacement

Cushing syndrome

- **Etiology:** Exogenous steroids, pituitary adenoma, ectopic ACTH, adrenal hyperplasia
- **Symptoms:** Moon facies, “buffalo hump,” hypertension, truncal obesity, depression, striae, diabetes, osteopenia, hypokalemia, metabolic acidosis
- **Diagnosis:** Check chemistry panel for acidosis and abnormalities of electrolytes; conduct dexamethasone suppression test or 24-hour urine free cortisol level
- **Treatment:** For Cushing syndrome, adenoma resection; if ectopic ACTH release, treat underlying neoplasm

Pituitary Disorders

Hormones released from pituitary gland include: ACTH, thyroid-stimulating hormone (TSH), luteinizing hormone/follicle stimulating hormone (LH/FSH), growth hormone (GH), prolactin.

Hypopituitarism

- **Etiology:** Invasive disease, infiltrative disease, infarction, head trauma, iatrogenic infection
- **Symptoms:** Depends on the hormone deficiency; GH, LH/FSH, TSH, ACTH, antidiuretic hormone
- **Diagnosis:** Blood test for specific hormones suspected in hypopituitarism
- **Treatment:** Treat the underlying cause and correct hormone deficiencies with appropriate oral/nasal hormonal medications

Hyperpituitarism

- **Etiology:** Adenoma, prolactinoma
- **Symptoms:** Headache, vision changes, additional symptoms specific to hormone released from pituitary gland
 - Prolactinoma has additional symptoms: Galactorrhea, amenorrhea, impotence
- **Diagnosis:** MRI, visual field testing for bitemporal hemianopsia (other defects may occur with larger lesions)
- **Treatment:** Surgical removal of adenoma, dopamine agonists for prolactinomas

Hypercalcemia

- **Symptoms**
 - “Moans” (stupor, depression, psychosis)
 - “Groans” (nausea, vomiting, constipation)
 - “Stones” (kidney stones, nephrogenic diabetes insipidus)
 - “Bones” (arthritis, fractures)
 - Other symptoms: Weakness, hypertonia, bradycardia
- **Etiology:** Malignancy (parathyroid hormone-related protein [PTHrP], local osteolysis), granulomatous disorders, Paget disease
- **Diagnosis:** Check parathyroid hormone, ionized calcium
- **Treatment:** Normal saline infusion for urinary excretion
 - If calcium is still elevated after normal saline infusion, consider diuretics to inhibit calcium reabsorption and bisphosphonates or calcitonin when hypercalcemia is secondary to malignancy
 - Glucocorticoids may be used to treat hypercalcemia in patients with multiple myeloma

Hypocalcemia

- **Symptoms:** Neuromuscular excitability (seizures, tetany), Chvostek sign, Trousseau sign, prolonged QT interval
- **Etiology:** Hypoparathyroidism, parathyroid hormone resistance, vitamin D deficiency
- **Diagnosis:** Check ionized calcium, parathyroid hormone values, renal function
- **Treatment:** Intravenous calcium drip acutely, oral calcium (calcitriol, if needed) chronically

Autoimmune Disease

Sarcoidosis

- **Definition:** Autoimmune disease causing noncaseating granulomas
- **Etiology:** Unknown
- **Symptoms:** Nonspecific symptoms such as fever, weight loss, arthralgias
- **Diagnosis:** Biopsy of granulomas, exclusion of other diseases, chest radiograph or computed tomography (CT) scan
 - Lip biopsy: Minor salivary glands will show noncaseating granulomas even with normal-appearing mucosal tissue
- **Treatment:** Systemic steroids

Rheumatoid Arthritis

- **Symptoms:** Morning pain and stiffness, erythema and warmth in joint, symmetric pattern, presence of rheumatoid nodules, ulnar deviation of metacarpophalangeal joints, swelling of the proximal interphalangeal joints, swan neck deformity, boutonnière deformity
- **Diagnosis:** Rheumatoid factor titers, classic radiologic findings, elevated erythrocyte sedimentation rate and C-reactive protein, anti-citrullinated protein antibody present
 - Can be associated with serositis, ocular disease, amyloidosis, atherosclerosis
 - Firm diagnosis requires at least 6 points from criteria for rheumatoid arthritis classification

Criteria for rheumatoid arthritis classification

	Description	Points
Joint involvement	1 large joint	0
	2–10 large joints	1
	1–3 small joints	2
	4–10 small joints	3
	> 10 joints	5
Serology	–RF and –ACPA	0
	+RF or low +ACPA	2
	+RF or high +ACPA	3
Acute phase reactants	Normal CRP and ESR	0
	Elevated CRP or ESR	1
Duration	< 6 weeks	0
	≥ 6 weeks	1

RF, rheumatoid factor; ACPA, anti-citrullinated protein antibody; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate.

Rheumatoid arthritis versus osteoarthritis

	Rheumatoid arthritis	Osteoarthritis
Systemic symptoms	Fatigue, stiffness, dull pain, inflammation	None
Disease process	Autoimmune	Degenerative
Sex	Women > Men	Women = Men
Effusion	Present	Rare
Nodular growth	Swan neck/boutonnière deformity	Heberden/Bouchard nodes
Joints affected	Symmetric involvement	Asymmetric involvement
Age	Middle age	Older age
Movement effect	Movement improves pain	Movement increases pain

- **Treatment:** Disease-modifying antirheumatic drugs such as methotrexate or a tumor necrosis factor alpha (TNF-alpha) inhibitor

Systemic Lupus Erythematosus

- **Symptoms:** Arthralgias, photosensitive rash (malar region is common), oral ulcers, pancytopenia, serositis
- **Diagnosis:** Anti-double standard DNA (anti-dsDNA), antinuclear antibody (ANA) testing, anti-Smith antibodies, antiphospholipid antibodies
- **Treatment**
 - Sun avoidance/protection, nonsteroidal anti-inflammatory drugs (NSAIDs)
 - Increasing doses of systemic steroids based on disease severity
 - Severe disease requires IV chemotherapeutic agents such as cyclophosphamide, azathioprine, rituximab

Myasthenia Gravis

- **Definition:** Autoimmune disorder with auto-antibodies to acetylcholine receptor
- **Symptoms:** Fluctuating, fatigable weakness classically affecting the eye muscles causing ptosis, extraocular muscle palsies, easy fatigability of proximal muscles, preserved deep tendon reflexes
- **Diagnosis:** Anti-nicotinic acetylcholine receptor (anti-nAChR) antibodies, anti-muscle-specific tyrosine kinase (anti-MuSK) antibodies, Tensilon (Hoffmann-LaRoche) test, electromyography (EMG)
- **Treatment**
 - Anticholinesterase inhibitors (ie, physostigmine)
 - Immunomodulators
 - Thymectomy; thought to reduce autoantibody production
- **Complication:** Myasthenic crisis leading to respiratory failure
 - Treat with IV immunoglobulin/plasmapheresis
 - May require temporary intubation

Sclerosing Syndromes

Disease	Definition	Symptoms	Diagnosis	Treatment
Scleroderma	Collagen overproduction	<ul style="list-style-type: none">• Skin tightening• GERD• Malabsorption• Raynaud disease	Skin biopsy	<ul style="list-style-type: none">• Vasodilatory medicine• NSAIDs• Steroids• Immune suppressants
Progressive systemic sclerosis	Disorder of connective tissue with thickening of dermal collagen bundles and fibrosis and vascular abnormalities in internal organs	<ul style="list-style-type: none">• Vasomotor disturbance• Fibrosis• Skin atrophy• Muscle atrophy• Internal organ dysfunction	<ul style="list-style-type: none">• Pruritis• Raynaud disease• Swallowing difficulty• Nausea, vomiting• Palpitations, cough, dyspnea• Hypertension• Joint pain and weakness	Immunomodulatory agents
Sjögren syndrome	Systemic autoimmune disease in which immune system attacks exocrine glands	<ul style="list-style-type: none">• Dry mouth• Dry eyes• Generalized dryness of mucosa	<ul style="list-style-type: none">• ANA, RF, anti-SSA/B autoantibodies• Lip biopsy• Eye exam, Schirmer test• Sialography	Steroids
Fibromyalgia	Widespread musculoskeletal pain, fatigue, depression, sleep disorder	Dull ache typically starting in muscles with trigger points	<ul style="list-style-type: none">• Widespread pain for > 3 months• Minimum of 11 trigger points• Exclude other comorbidities• Check CBC, ESR, thyroid function	<ul style="list-style-type: none">• Pain control• Pregabalin• Physical therapy• Stress management

GERD, gastroesophageal reflux disease; RF, rheumatoid factor; anti-SSA/B, anti-Sjögren syndrome A/B; CBC, complete blood count; ESR, erythrocyte sedimentation rate.

Allergy

	Type 1	Type 2	Type 3	Type 4
Mechanism	IgE	Antibody-mediated cytotoxic reactions	Immune complex reaction activating complement	Delayed hypersensitivity activating T cells
Example	Anaphylaxis	Penicillin-induced hemolytic anemia	Serum sickness	Poison ivy dermatitis

IgE, immunoglobulin E.

Vasculitides

Disease	Symptoms	Diagnosis	Pathology	Treatment
Wegener granulomatosis	<ul style="list-style-type: none"> • Fever • Nosebleeds • Persistent nasal congestion • Muscle aches • Fatigue 	<ul style="list-style-type: none"> • Elevated ESR • Positive c-ANCA • Pulmonary nodules in chest radiograph 	<ul style="list-style-type: none"> • Necrotizing granulomas in small/medium vessels • Nodules in upper and lower respiratory tract 	Chemotherapy
Churg-Strauss angiitis	<ul style="list-style-type: none"> • Asthma • Neuropathy • Fever • Rash • Myalgias • Angina • Cough 	<ul style="list-style-type: none"> • Positive p-ANCA • Chest radiograph with changing pulmonary infiltrates 	<ul style="list-style-type: none"> • Eosinophil and granulomatous inflammation of the respiratory tract with necrotizing vasculitis of small- and medium-sized vessels 	Steroids
Microscopic polyangiitis	<ul style="list-style-type: none"> • Kidney inflammation • Weight loss • Skin lesions • Nerve damage • Fevers 	<ul style="list-style-type: none"> • Elevated ESR • Positive p-ANCA 	<ul style="list-style-type: none"> • Nongranulomatous vasculitis in small vessels of lung, kidneys, and nerves 	Steroids
Giant cell arteritis	<ul style="list-style-type: none"> • Severe headaches • Temporal artery tenderness • Jaw claudication 	<ul style="list-style-type: none"> • Elevated ESR • New-onset headaches • Tender and pulseless temporal artery • Biopsy of temporal artery shows giant cells and infiltration with mononuclear cells 	<ul style="list-style-type: none"> • Granulomas of large- and medium-sized branches of the carotid artery 	High-dose steroids
Takayasu arteritis	<ul style="list-style-type: none"> • Limb/organ ischemia • Hypertension • Systolic blood pressure changes on each limb 	<ul style="list-style-type: none"> • Elevated ESR • Angiography 	<ul style="list-style-type: none"> • Pulseless vasculitis of large branches of the aorta 	<ul style="list-style-type: none"> • Steroids • Blood pressure control
Polyarteritis nodosa	<ul style="list-style-type: none"> • Rash • Abdominal pain • Neuropathies • Fever • Muscle/joint pain 	<ul style="list-style-type: none"> • Elevated ESR • Positive p-ANCA • Arteries have aneurysmal dilations 	<ul style="list-style-type: none"> • Small/medium vessel necrotizing disease of kidney, nerves, GI tract, and brain 	<ul style="list-style-type: none"> • Steroids • Chemotherapy
Behçet disease	<ul style="list-style-type: none"> • Oral and genital ulcerations • Erythema nodosum 	Positive pathergy test	Unknown etiology	Steroids

ESR, erythrocyte sedimentation rate; c-ANCA, cytoplasmic antineutrophil cytoplasmic antibodies; p-ANCA, perinuclear antineutrophil cytoplasmic antibodies.

Serology Tests

	Disease association
ANCA	Wegener granulomatosis
Anti-dsDNA	Lupus erythematosus
ANA	<ul style="list-style-type: none"> • Rheumatoid arthritis • Lupus erythematosus • Sjögren syndrome • Diffuse and limited scleroderma • Myositis • Wegener granulomatosis
Anti-SSA	<ul style="list-style-type: none"> • Sjögren syndrome • Lupus erythematosus
Anti-SSB	<ul style="list-style-type: none"> • Sjögren syndrome • Lupus erythematosus
RF	<ul style="list-style-type: none"> • Rheumatoid arthritis • Lupus erythematosus • Sjögren syndrome • Diffuse and limited scleroderma • Myositis • Wegener granulomatosis
ANCA, antineutrophil cytoplasmic antibodies; anti-SSA, anti-Sjögren syndrome A; anti-SSB, anti-Sjögren syndrome B; RF, rheumatoid factor.	

Neurologic Disorders

Syncope

Transient loss of consciousness and postural tone.

- **Symptoms:** Paleness, lightheadedness, sweating, nausea
- **Diagnosis:** History and physical examination with vital signs, ECG, Holter monitoring, tilt-table testing
- **Treatment:** Treat underlying condition (always initially search for cardiac etiology)

Cardiogenic	Neurogenic	Vasovagal	Orthostatic
<ul style="list-style-type: none"> • Aortic dissection • Arrhythmias • Valvular heart disease • Pulmonary embolus • Atrial myxoma • Myocardial infarction 	<ul style="list-style-type: none"> • Inadequate supply of oxygenated blood to the brain • Autonomic insufficiency • Carotid sinus hypersensitivity • Seizures 	<ul style="list-style-type: none"> • Straining • Uneasy situations • Blood draws 	<ul style="list-style-type: none"> • Elderly • Dehydrated patients • Patients on antihypertensive medications

Seizure

Abnormal discharge of cortical neurons.

Primary generalized seizures

- Tonic-clonic: Associated with urinary incontinence, postictal confusion
- Myoclonic: Abrupt contraction of a single muscle group
- **Diagnosis:** Lab tests to evaluate electrolytes, brain magnetic resonance imaging (MRI), electroencephalogram (EEG)
- **Treatment:** Broad-spectrum anticonvulsants

Focal seizures

- Simple: No loss of consciousness but symptoms of twitching/jerking
- Complex focal: Impairment of consciousness develops, causing automatisms
- Complex focal with secondary generalization: Spreads to involve the cerebral cortex
- **Diagnosis:** EEG, brain imaging
- **Treatment:** Anticonvulsants, surgery

Static epileptus

- Continuous seizure activity > 30 minutes or recurrent seizures without return of consciousness
- **Treatment:** Check airway, breathing, circulation; draw blood for lab tests; consider thiamine, glucose, benzodiazepines, phenytoin

Cerebrovascular Accident

Acute onset focal neurologic changes secondary to blood flow disruption to the brain.

Ischemic stroke

Emboli from internal carotid artery or heart causing blood vessel blockage.

- **Diagnosis:** Noncontrast head CT scan
- **Treatment:** Tissue plasminogen activator if < 3 hours from stroke symptom onset, antiplatelet medications, statins, blood pressure control

Hemorrhagic stroke

Associated with headache and rapid loss of consciousness, with hypertension being primary cause.

- **Diagnosis:** Noncontrast head CT scan
- **Treatment:** Supportive care, endovascular procedures, open surgery to stop bleeding

Intracranial Bleeding

	Epidural hematoma	Subdural hematoma	Subarachnoid hematoma
Vessels	Middle meningeal artery	Bridging veins	Aneurysmal bleeding
Symptoms	Waxing and waning consciousness	Progressive decline of mental status	"Worst headache of my life"
Diagnosis	Lens-shaped hematoma on CT scan	Crescent-shaped hematoma on CT scan	Xanthochromia on lumbar puncture
Treatment	Decompression	Decompression	Coiling or clipping

Declined Mental Ability

	Dementia	Delirium
Definition	Acquired syndrome with slow decline in memory and cognition	Rapid-onset confusion and disorientation with fluctuating intensity
Etiology	Alzheimer, Lewy body dementia, medications, metabolic disorders, vascular infarction, chronic disease	Elderly hospitalized patients with risk factors including infection, depression, fever
Symptoms	Personality changes, trouble with activities of daily living, impaired judgment	Waxing and waning alterations in consciousness and cognition
Diagnosis	Mini-mental status exam, CBC, electrolytes, creatinine, liver function tests, TSH/B12 levels, rapid plasmin regain test, HIV test	Physical exam with same lab tests as for dementia
Treatment	Treat underlying cause, antidepressants, antipsychotics	Behavioral and environmental change, low-dose haloperidol if needed
CBC, complete blood count.		

Neuroleptic Malignant Syndrome

- **Etiology:** Decreased levels of dopamine activity secondary to dopamine receptor blockade
- **Symptoms:** Increased body temperature, altered consciousness, diaphoresis, rigid muscles, autonomic imbalance
- **Treatment:** Stop neuroleptic drugs, treat hyperthermia. intensive care unit care, dantrolene for muscle rigidity, intravenous fluids

Serotonin Syndrome

- **Etiology:** Excess of serotonergic activity in the central nervous system and peripheral serotonin receptors from therapeutic drug use, recreational drug use, or drug interactions
- **Symptoms:** Headache, agitation, confusion, shivering, sweating, hyperthermia, hypertension, tachycardia, nausea, vomiting, hyper-reflexia, tremors, muscle twitching
- **Treatment:** Stop drugs, give serotonin antagonists, supportive care for sympathetic hypersensitivity and concomitant symptoms

Differentiating Serotonin Syndrome from Neuroleptic Malignant Syndrome

	Serotonin syndrome	Neuroleptic malignant syndrome
Onset	24 hours	Days to weeks
Causative agent	Serotonin agonist	Dopamine antagonist
Muscular findings	Excitability	Rigidity
Treatment	Serotonin antagonist and benzodiazepines	Dopamine agonist and dantrolene
Resolution	Within 24 hours	Days to weeks

Alcohol Withdrawal

Alcohol is a central nervous system depressant that enhances gamma-aminobutyric acid (GABA) inhibitory tone and inhibits excitatory amino acid activity. Abrupt removal of alcohol results in overactivity of the central nervous system.

- **Symptoms:** Anxiety 2 to 3 days after last drink; sympathetic hyperactivity and hypersensitivity; delirium tremens 36 hours after last drink
- Delirium tremens: Hypertension, tachycardia, agitation, hyperthermia

Clinical Institute Withdrawal Assessment for Alcohol (CIWA) protocol

To assess the level of withdrawal and medication needed, use the CIWA protocol. The CIWA protocol has 10 categories noted below, with scoring of 0 to 7 in all categories except orientation, which is scored 0 to 4.

- Nausea/vomiting
- Anxiety
- Visual disturbance
- Paroxysmal sweats
- Tactile disturbance
- Orientation
- Auditory disturbance
- Headache
- Agitation
- Tremors

Score	Withdrawal
Less than 8	Prophylaxis
8–15	Mild
16–25	Moderate
> 25	Severe

General treatment

- Hydration
- Correct electrolyte imbalance
- Thiamine, 100 mg IV daily for 3 days, then orally daily
- Folic acid, 1 mg orally daily
- Benzodiazepine taper (long-acting preferred for smoother withdrawal course and smaller chance of seizures or recurrent withdrawal symptoms)

Parkinson Disease

Progressive, idiopathic disease.

- **Symptoms:** Resting tremor, bradykinesia, cogwheel rigidity, postural instability; symptoms begin asymmetrically
- **Pathology:** Affects the dopamine neurons of the substantia nigra
- **Treatment:** Levodopa/carbidopa, dopamine agonists, anticholinergics

Huntington Disease

Progressive, autosomal dominant disease (cytosine-adenine-guanine expansion in DNA).

- **Symptoms:** Chorea, dementia, psychiatric symptoms
- **Pathology:** Atrophy of the caudate nucleus
- **Treatment:** None available

Alzheimer Disease

- **Etiology:** Unknown but genetic component may be present; low levels of acetylcholine
- **Pathology:** Senile plaques with central amyloid core and bundles of neurofilaments in neuronal cytoplasm
- **Symptoms:** Mild forgetfulness with poor concentration and changes in personality that worsen in later stages to paranoid delusions, hallucinations, and need for assistance in all activities of daily living
- **Treatment:** Avoid anticholinergics; donepezil is a first-line agent.

Lewy Body Dementia

- **Etiology:** Loss of cholinergic neurons and death of dopaminergic neurons
- **Pathology:** Lewy bodies (alpha synuclein cytoplasmic inclusions) present in the cortex
- **Symptoms:** Variation in cognition and attention; recurrent hallucinations and Parkinson-like motor symptoms
- **Treatment:** Donepezil for cognition and levodopa for motor symptoms; otherwise, palliation

Perioperative Management

Shock

Pathophysiologic state associated with decreased tissue perfusion and hypoxia.

Categories

	Cardiogenic	Hypovolemic	Distributive
Etiology	Myocardial infarction	Trauma and bleeding (see classes of hemorrhage, page 42)	Sepsis, anaphylactic, neurogenic
Cardiac output	Decreased	Decreased	Increased
Systemic vascular resistance	Increased	Increased	Decreased
Jugular venous pressure	Increased	Flat	N/A
Extremities	Cool, clammy	Cool, clammy	Warm

Stages of shock

	Reversible	Definition
Initial	Yes	No signs of shock but cells starting to use anaerobic metabolism
Compensatory	Yes	Compensatory mechanisms try to reverse the symptoms of shock (eg, hyperventilation to correct acidosis)
Progressive	No	Compensatory mechanisms cannot correct shock symptoms, leading to worsening acidosis and decreased organ perfusion
Refractory	No	Organ failure and death

Treatment

- Aggressive IV fluid hydration with initial 2 L bolus (except in cardiogenic shock)
- Cardiogenic shock: Decrease afterload, increase cardiac output, decrease myocardial oxygen demand
- Treat underlying cause

Classes of hemorrhage

	Blood loss	Pulse	Blood pressure	Respiratory rate	Urine output	Orientation	Treatment
1	< 15%	Normal	Normal	Normal	Normal	Normal	Minimal
2	15%–30%	> 100 BPM	Normal	Mild tachypnea	20–30 cc/hr	Anxious	IV fluids
3	30%–40%	> 120 BPM and weak	Decreased	Marked tachypnea	20 cc/hr	Confused	Aggressive fluids and packed RBCs
4	> 40%	> 140 BPM and nonpalpable	Marked decrease	Marked tachypnea	Negligible	Comatose	Aggressive therapy

BPM, beats per minute.

Postoperative Fever

Postoperative day	Cause
1–2	Wind (pneumonia)
3–5	Water (urinary tract infection)
4–6	Walking (deep vein thrombosis/pulmonary embolism)
5–7	Wound (surgical site infection)
> 7	Wonder drugs (drug fever)

Treatment

- Physical examination
- Complete blood count (CBC) with differential count
- Panculture the patient
- Replace all lines
- Review old culture results
- Lower extremity Doppler study
- Chest radiograph

Common Perioperative Issues

Fluid management	<p>Maintenance</p> <ul style="list-style-type: none">• Follow the 4/2/1 rule for maintenance fluid management<ul style="list-style-type: none">– 4 cc/kg for the first 10 kg– 2 cc/kg for the second 10 kg– 1 cc/kg for each kg thereafter• Additional fluid infusion for instances such as<ul style="list-style-type: none">– Fever– Drains– Gastrointestinal losses– Burns• Decreased fluid infusion for instances such as<ul style="list-style-type: none">– Edematous states– Hypothyroidism– Renal failure <p>Hypovolemia</p> <ul style="list-style-type: none">• Colloid versus crystalloid therapy – Crystalloid therapy is the gold standard except in cases of severe blood loss; begin therapy with 2 liters of isotonic crystalloid fluid<ul style="list-style-type: none">– Colloid therapy is more expensive and has not shown a benefit over crystalloid therapy• Blood transfusion<ul style="list-style-type: none">– Use in cases of severe hemorrhagic hypovolemia or hemorrhagic shock <p>Postoperative fluid overload</p> <ul style="list-style-type: none">• Signs include pitting edema, hypertension, lung crackles, shortness of breath, increased jugular vein distention• Treatment<ul style="list-style-type: none">– Fluid restriction– Sodium restriction– Diuretic therapy– Monitor urine output– Reposition patient to decrease dependent collection of fluids
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(Common Perioperative Issues cont)

**Cardiovascular
management**

This is discussed in detail in the cardiovascular section (see page 2).

Beta blockers

- High-risk patients may benefit from preoperative beta blockade, but careful monitoring for appropriate heart rate and blood pressure is critical
- Choice of beta blocker should be discussed with cardiologist or medical consult team; usually selective beta-1 blockade is preferred

Hypertension

- Ensure patient is taking preoperative medications
- Look for other causes (pain, bladder distension, hypoxia, hypervolemia)
- For new onset postoperative hypertension without an underlying cause, consider nitro-prusside or labetalol and speak to cardiologist regarding further management

Atrial fibrillation

- Rule out thyroid disease, systemic illness, pulmonary embolus, and acute myocardial infarction
- Symptoms include palpitations, dizziness, irregularly irregular pulse
- ECG will show a wavy baseline with loss of P waves
- Acute-onset atrial fibrillation in an unstable patient requires immediate electrical cardioversion
- Acute-onset atrial fibrillation in a stable patient requires rate control with beta blockers and calcium channel blockers
 - Once rate control is achieved, cardioversion is required to convert patient back to normal sinus rhythm
 - If the arrhythmia has been present for more than 48 hours, anticoagulation is required for 3 weeks before and 4 weeks after cardioversion
 - Earlier cardioversion is done if no thrombus is present on transesophageal echocardiogram

Chest pain

- ECG (ST elevations, T wave changes, Q waves for acute myocardial infarction diagnosis)
- Chest radiograph
- Test for troponins and creatine kinase-MB (will elevate 6–8 hours post chest pain onset)
- Supplemental oxygen
- Speak to cardiologist regarding therapy for ischemic heart disease
 - Beta blockers
 - Aspirin
 - Nitroglycerin
 - Heparin therapy
- Cardiac catheterization for high-risk patients (STEMI)
- Cardiac stress testing for low-risk patients

(Common Perioperative Issues cont)

Pulmonary management	<p>Acute respiratory distress syndrome</p> <ul style="list-style-type: none">• Hypoxemic respiratory failure with bilateral lung infiltrates• No evidence of heart failure – Symptoms include tachypnea and diffuse lung crackles – Chest radiograph will show bilateral alveolar infiltrates – Pulmonary wedge pressure is less than 18 mm Hg – Partial pressure of oxygen (PaO₂)/fractional inspired oxygen (FiO₂) is less than 200 mm Hg<ul style="list-style-type: none">– If PaO₂/FiO₂ is between 200 and 300 mm Hg, diagnosis is acute lung injury• Treatment<ul style="list-style-type: none">– Treat underlying cause– Low tidal volumes (6 cc/kg)– Low PEEP– Conservative fluid management– Plateau pressure less than 30 cm H₂O <p>Postoperative respiratory failure</p> <ul style="list-style-type: none">• Asthma<ul style="list-style-type: none">– Supplemental oxygen– Short-acting beta agonist– Systemic steroids– IV magnesium sulfate• Pulmonary embolus<ul style="list-style-type: none">– Supplemental oxygen– Heparin therapy• Guillain-Barré syndrome<ul style="list-style-type: none">– Supplemental oxygen– Mechanical ventilation– Plasmapheresis or IV immunoglobulin infusion• Pneumonia<ul style="list-style-type: none">– Culture sputum– Empiric therapy with Unasyn (Pfizer) or Zosyn (Pfizer)
Epilepsy	<p>Postoperative status epilepticus</p> <ul style="list-style-type: none">• Prolonged and sustained loss of consciousness• Persistent convulsions• Look for organic cause and rule out metabolic disorder, neoplasm, intracranial infection, stroke, drug intoxication• Treatment<ul style="list-style-type: none">– Establish airway– Establish IV access– STAT labs (CBC, electrolytes, liver panel, anticonvulsant medication level)– IV benzodiazepines (diazepam)– Loading dose of anticonvulsant medication<ul style="list-style-type: none">◦ IV dextrose◦ IV thiamine

(Common Perioperative Issues cont)

Diabetes management	<p>Patient on insulin</p> <ul style="list-style-type: none">• Preoperative: Give 50% long-acting insulin dose on morning of surgery; start IV glucose drip• Postoperative: Start IV glucose drip <p>Patient not on insulin</p> <ul style="list-style-type: none">• Preoperative: Discontinue oral hypoglycemics and metformin 24 hours preoperatively• Postoperative: Insulin drip with a short-acting sliding scale																												
Steroid management	<p>Patients taking steroids chronically</p> <ul style="list-style-type: none">• Daily dose of 20 mg or more of prednisone or equivalent• More than 3 weeks of steroid treatment• An acute cortisol deficiency secondary to surgical stress, in a patient with adrenal insufficiency, will lead to adrenal crisis; symptoms include headache, nausea, vomiting, shock, and confusion; without steroid dosing, this can be fatal <p>Treatment: Steroid stress dosing</p> <table><tr><th></th><th>Preoperatively</th><th>Postoperatively</th></tr><tr><td>Minor surgery</td><td>Normal AM dose</td><td>Normal dose</td></tr><tr><td>Moderate surgery</td><td>Normal AM dose plus 50 mg IV hydrocortisone</td><td>Normal AM dose plus 25 mg IV hydrocortisone every 8 hours for 24 hours</td></tr><tr><td>Major surgery</td><td>Normal dose plus 100 mg hydrocortisone</td><td>Normal dose plus 25 mg hydrocortisone every 8 hours for 24 hours, then taper to normal dose</td></tr><tr><td colspan="3">AM, morning.</td></tr></table>		Preoperatively	Postoperatively	Minor surgery	Normal AM dose	Normal dose	Moderate surgery	Normal AM dose plus 50 mg IV hydrocortisone	Normal AM dose plus 25 mg IV hydrocortisone every 8 hours for 24 hours	Major surgery	Normal dose plus 100 mg hydrocortisone	Normal dose plus 25 mg hydrocortisone every 8 hours for 24 hours, then taper to normal dose	AM, morning.															
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AM, morning.																													
Antibiotic prophylaxis	<p>Conditions that require prophylaxis for oral procedures</p> <ul style="list-style-type: none">• Prosthetic cardiac valve• Prosthetic material for cardiac valve repair• History of infective endocarditis• Congenital heart disease (CHD)<ul style="list-style-type: none">– Unrepaired cyanotic CHD<ul style="list-style-type: none">◦ Completely repaired congenital defect with prosthetic material or device within 6 months of repair– Repaired CHD with residual disease<ul style="list-style-type: none">◦ Cardiac transplant patients who have developed valvulopathy <table><tr><th>Mode</th><th>Agent</th><th>Dose: Adults</th><th>Dose: Children</th></tr><tr><td>Oral</td><td>Amoxicillin</td><td>2 g</td><td>50 mg/kg</td></tr><tr><td>IV/IM</td><td>Ampicillin</td><td>2 g</td><td>50 mg/kg</td></tr><tr><td>IV/IM</td><td>Cefazolin</td><td>1 g</td><td>50 mg/kg</td></tr><tr><td>Oral (penicillin allergy)</td><td>Clindamycin</td><td>600 mg</td><td>20 mg/kg</td></tr><tr><td>IV/IM (penicillin allergy)</td><td>Clindamycin</td><td>600 mg</td><td>20 mg/kg</td></tr><tr><td colspan="4">IM, intramuscular.</td></tr></table> <ul style="list-style-type: none">• No antibiotic prophylaxis is required for dialysis patients or patients who have had joint replacement or solid organ transplants unless otherwise specified by their doctors	Mode	Agent	Dose: Adults	Dose: Children	Oral	Amoxicillin	2 g	50 mg/kg	IV/IM	Ampicillin	2 g	50 mg/kg	IV/IM	Cefazolin	1 g	50 mg/kg	Oral (penicillin allergy)	Clindamycin	600 mg	20 mg/kg	IV/IM (penicillin allergy)	Clindamycin	600 mg	20 mg/kg	IM, intramuscular.			
Mode	Agent	Dose: Adults	Dose: Children																										
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Recommended Readings

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Anesthesia

Jason Jamali and Stuart Lieblich

- ▶ Basic Patient Preanesthesia Management
- ▶ Pharmacology
- ▶ Mechanical Ventilation
- ▶ Perioperative Care
- ▶ Anesthesia for Special Populations

Basic Patient Preanesthesia Management

Preoperative Airway Evaluation

- Mandibular opening (> 3 finger)
- Dentition
- Thyromental distance (> 6 cm)
- Cervical spine (35 degrees of neck extension)
- Neck (scars from previous surgeries)
- Mallampati classification
 1. Soft/hard palate, uvula, tonsillar pillars visible
 2. Soft/hard palate, parts of uvula visible (tonsillar pillars not visible)
 3. Soft/hard palate, base of uvula visible
 4. Hard palate only visible
- Preoperative testing: Consider complete blood count (CBC), electrolytes, coagulation studies, pregnancy test, electrocardiogram (ECG) (age > 50 and/or general anesthesia), chest radiograph depending on the type of surgery

Preoperative Fasting Guidelines

Ingested material	Minimum fasting period (hours)
Clear liquids	2
Breast milk	4
Infant formula	6
Nonhuman milk	6
Light meal	6
Heavy meal	8+

American Society of Anesthesiologists (ASA) Classification

- | | |
|--|--|
| I: Normal healthy patient | V: Moribund patient not expected to survive without surgery |
| II: Mild-moderate systemic disease | VII: Declared brain dead, planned for organ harvest |
| III: Severe systemic disease | E: Any emergency |
| IV: Severe systemic disease that is a constant threat to life | |

Perioperative Monitoring

Pulse oximetry	<ul style="list-style-type: none">• Monitors oxygen saturation of hemoglobin and heart rate• Sources of error: Shivering, fingernail polish, carboxyhemoglobin, methemoglobin, methylene blue, hypothermia, hypotension, hypovolemia, hypoxia, ambient light
ECG	<ul style="list-style-type: none">• Used for identification of dysrhythmias, myocardial ischemia, pacemaker function• Leads II and V5 are commonly used—more sensitive to ischemia
Blood pressure	<ul style="list-style-type: none">• Noninvasive blood pressure monitoring reflects blood flow, whereas invasive monitoring is more directly correlated with blood pressure and allows for real-time continuous monitoring via placement of an intra-arterial catheter• Invasive blood pressure monitoring is indicated in patients that are hemodynamically unstable, undergoing major surgery where a higher blood loss is anticipated, receiving vasoactive medications, or requiring frequent lab draws• Due to gravity, blood pressure in the lower extremities may be higher• Source of error: Improper size cuff will give inaccurate results<ul style="list-style-type: none">– Too large of a cuff: Falsely low blood pressure– Too tight of a cuff: Falsely elevated blood pressure
Capnography	<ul style="list-style-type: none">• Carbon dioxide measurement allows for<ul style="list-style-type: none">– Assessment of ventilation– Assessment of circulation– Verification of intubation– Identification of anesthetic circuit malfunction (eg, leaks, disconnection)
Bispectral index (BIS)	<ul style="list-style-type: none">• Objectively measures the depth of anesthesia (intravenous or inhalational)• Mechanism – Uses electroencephalogram (EEG) data obtained via scalp electrodes to calculate a depth of anesthesia represented by a dimensionless number between 0 and 100<ul style="list-style-type: none">– Deeper levels of anesthesia are represented by lower numbers• Should be used only as an adjunct to evaluation as several sources of error exist<ul style="list-style-type: none">– Paradoxical changes with certain anesthetics (ketamine, N₂O)– Electrical device interference (electrocautery)– Certain clinical conditions (hypoglycemia, Alzheimer disease)

Anesthesia Techniques

Continuum of sedation*

	Minimal sedation	Moderate sedation	Deep sedation	General anesthesia
Comment	Anxiolysis	Conscious sedation; drug-induced depression of consciousness	Drug-induced depression of consciousness	Drug-induced loss of consciousness
Cognitive/physical coordination	Impaired			

(Continuum of sedation cont)*

	Minimal sedation	Moderate sedation	Deep sedation	General anesthesia
Responsiveness	Normal response to verbal commands	Purposeful response to verbal or tactile stimulation	Purposeful response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention needed	Intervention may be required	Intervention required
Spontaneous ventilation	Unaffected	Adequate	Inadequate	Inadequate
Cardiovascular function	Unaffected	Maintained	Maintained	Impaired

*American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists.
Practice guidelines for sedation and analgesia by non-anesthesiologists. Anesthesiology 2002;96:1004–1017.

Intubation techniques

Laryngeal mask airway (LMA)	<ul style="list-style-type: none">• A supraglottic airway device that may be used as an alternative to tracheal intubation during general anesthesia, especially for cases of short duration• May be used during airway emergencies when tracheal intubation has failed• Placement is performed blindly without the use of a laryngoscope and with the patient in the sniffing position• Disadvantage: Aspiration of gastric contents is not prevented• In addition to the classic LMA, other variants exist – Flexible LMA: Has wire reinforcement of the tube, allowing the stem to be moved out of the view of the surgical site<ul style="list-style-type: none">– Intubating LMA: Has a rigid, wider, and shorter airway stem to allow for intubation through the LMA– ProSeal LMA (Teleflex): Has a drainage tube that lies at the top of the esophagus; allows for better separation of the respiratory and gastrointestinal tracts
Endotracheal tube intubation	<ul style="list-style-type: none">• Used to establish a secure airway in patients during general anesthesia• Variations include: Armored tubes (metal wire reinforcement) and preformed tubes (oral and nasal Ring-Adair-Elwyn [RAE])• Advantages over LMA<ul style="list-style-type: none">– Protects against aspiration of gastric contents– May be used in positions other than supine– Higher positive airway pressures can be obtained– Better when the upper airway anatomy is altered
Tracheostomy	<ul style="list-style-type: none">• Surgical airway that allows direct access to the trachea via a neck incision; this may be performed percutaneously• Indicated for long-term ventilation and may be helpful with ventilator weaning
Cricothyrotomy	<ul style="list-style-type: none">• A temporary surgical airway obtained through the cricothyroid membrane via a neck incision• Used during airway emergencies when other nonsurgical attempts at securing the airway have failed

Pharmacology

Basic Pharmacokinetics

The pharmacokinetics of a drug is a function of the volume of distribution, drug clearance, and patient-specific factors (age, disease, nutrition, hydration, etc).

Volume of distribution	<ul style="list-style-type: none"> • Dose of drug administered/concentration of drug in plasma • Decreased by high protein binding affinity, ionization, decreased lipid solubility
Clearance	<ul style="list-style-type: none"> • Volume of plasma cleared of drug in mL per minute as a result of renal elimination and metabolism (liver and other tissues: kidney, lung, gastrointestinal tract) • Renal elimination is improved with increased water solubility and inhibited by protein binding and lipid solubility • Elimination half-time: Time required to decrease drug concentration by 50% (~5 half-times required for total elimination)
Redistribution	<ul style="list-style-type: none"> • Initially drugs preferentially distribute to highly perfused tissues (eg, brain, heart, kidneys) • Eventually a concentration gradient is developed that allows transfer of drug to the remaining less perfused tissues (fat, skeletal muscle)
First-pass hepatic effect	<ul style="list-style-type: none"> • Oral drugs are absorbed by the gastrointestinal tract and pass through the liver via the portal circulation before entering the systemic circulation • Drugs may be variably metabolized during this process

Volatile Agents

- Concentration of volatile agent is proportional to its partial pressure
- For volatile agents, the goal is to maintain a specific partial pressure in the brain tissue; the partial pressure of volatile agent in the brain is in equilibrium with the blood and alveoli (pulmonary artery pressure [PAP])
- Since PAP can be measured, it is used to determine anesthetic depth
- PAP is a function of the following
 - Fractional inspired oxygen (FiO_2)
 - Alveolar ventilation
 - Anesthetic circuit
 - Blood gas coefficient
 - Cardiac output
 - Solubility of the anesthetic in blood/tissue
 - Alveolar-venous partial pressure difference
- The minimum alveolar concentration (MAC) is the concentration (partial pressure) of volatile anesthetic (at 1 atm and measured during steady state) that prevents movement in 50% of patients during a surgical stimulus
 - The higher the MAC, the less the potency

Comparison of volatile agents

	Advantages	Disadvantages	MAC
Sevoflurane	<ul style="list-style-type: none"> • Rapid induction/recovery • Minimal airway irritation 	Potential renal concerns (compound A)	2.05
Isoflurane	<ul style="list-style-type: none"> • Relatively slower onset of action • Pungent odor 	Coronary steal effect	1.15
Desflurane	Rapid induction/recovery	Airway irritation	6.0
Nitrous oxide	<ul style="list-style-type: none"> • Analgesia • Second gas effect • Safe in malignant hypothermia-susceptible patients • Rapid induction/recovery 	<ul style="list-style-type: none"> • Expansion of air-filled spaces • Higher combustion risk • Postoperative nausea and vomiting • Megaloblastic anemia • Chronic use may result in peripheral neuropathy • Possible teratogen 	105

Systemic effects of volatile agents

For each agent, the effects are generally dose dependent.

Blood pressure	(+/-0)	Nitrous oxide
	(-)	Halothane, isoflurane, desflurane, sevoflurane
Heart rate	(+)	Isoflurane, desflurane, sevoflurane (only if MAC > 1.5)
Cardiac contractility	(-)	All of the volatile agents result in decreased contractility, although nitrous oxide appears to cause relatively less myocardial depression
Systemic vascular resistance	(-)	Isoflurane, desflurane, sevoflurane
	(0)	Halothane
Ventilation	Most volatile anesthetics (except isoflurane) <ul style="list-style-type: none"> • Increase respiratory rate • Decrease tidal volume • Decrease minute ventilation • Produce a high carbon dioxide level 	

Opioids

Examples		<ul style="list-style-type: none">• Phenylpiperidines: Meperidine, fentanyl, sufentanil, remifentanil• Diphenylheptanes: Methadone, propoxyphene• Morphine group: Morphine, codeine, hydrocodone, oxycodone, oxymorphone, hydromorphone, nalbuphine, butorphanol, levorphanol, pentazocine
Mechanism of action		<ul style="list-style-type: none">• Agonist at various endogenous opioid receptors (mu1, mu2, delta, kappa, sigma) within the nociceptive pathways• Involves decreased presynaptic release of acetylcholine and substance P
Effects	CNS	Analgesia, sedation, decreased cerebral blood flow, miosis
	CV	Dose-dependent bradycardia (meperidine is an exception—myocardial stimulation)
	Respiratory	Dose-dependent respiratory depression, antitussive
	GI	Nausea, vomiting, decreased motility
	GU	Urinary retention
Concerns		<ul style="list-style-type: none">• Tolerance and dependence• Chest wall rigidity• Crosses placenta• Histamine release with morphine and meperidine
Reversal		<ul style="list-style-type: none">• Naloxone<ul style="list-style-type: none">– Adult dose: 0.04 to 0.4 mg IV; repeat dose or increase dose to 2 mg if no response– Pediatric dose: 0.001 to 0.005 mg/kg IV
Allergy		<ul style="list-style-type: none">• Flushing, hives, pruritus, diaphoresis, dysphagia, facial/airway swelling, anaphylaxis• Pseudo-allergy may produce mild symptoms secondary to histamine release
CNS, central nervous system; CV, cardiovascular; GI, gastrointestinal; GU, genitourinary; IV, intravenously.		

Intravenous Medications

Barbiturates

Example	Pentobarbital, methohexital
Mechanism of action	GABA potentiation, direct action on GABA _A receptor chloride channels, some action on calcium ion channels
Dosage	<ul style="list-style-type: none">• Induction: 1 to 1.5 mg/kg• Sedation: 0.75 to 1 mg/kg followed by 0.5 mg/kg every 2 to 5 minutes

(Barbiturates cont)

Indications		<ul style="list-style-type: none"> • Sedation • Induction of anesthesia • Anesthesia
Effects	CNS	Decreases cerebral blood flow (CBF) and intracranial pressure (ICP) (methohexital may increase epileptic foci)
	CV	Decreases blood pressure via peripheral vasodilation (transient if intact baroreceptor reflex)
	Respiratory	<ul style="list-style-type: none"> • Depression of ventilation/apnea • Minimal depression of laryngeal/cough reflexes—may predispose to laryngospasm
GABA, gamma-aminobutyric acid; CNS, central nervous system; CV, cardiovascular.		

Propofol

Mechanism of action		<ul style="list-style-type: none"> • Primarily via potentiation of the GABA_A receptors • Depresses reticular activating system
Pharmacokinetics		High lipid solubility, rapid redistribution, hepatic metabolism
Dosage		<ul style="list-style-type: none"> • Induction: 2 to 2.5 mg/kg • Sedation: 50 to 100 mcg/kg per minute
Indications		<ul style="list-style-type: none"> • Sedation • Induction • General anesthesia
Effects	CNS	<ul style="list-style-type: none"> • Decreases CBF and ICP • Antiemetic • Anticonvulsant
	CV	<ul style="list-style-type: none"> • Decreases systemic vascular resistance • Negative inotropism
	Respiratory	<ul style="list-style-type: none"> • Dose-dependent ventilatory depression • Apnea • Bronchodilation
	GI	Antiemetic
	Local	<ul style="list-style-type: none"> • Pain on injection • Severe tissue damage associated with intra-arterial injections and infiltration
Contraindications		<ul style="list-style-type: none"> • Lipid disorders • Acute intermittent porphyria • Avoid if egg, soy, or peanut allergy are present
GABA, gamma-aminobutyric acid; CNS, central nervous system; CV, cardiovascular; GI, gastrointestinal.		

Ketamine

Mechanism of action		Various receptors (NMDA agonist, opioid, monoaminergic, muscarinic, calcium ion channels)
Pharmacokinetics		<ul style="list-style-type: none">• High lipid solubility: Crosses blood-brain barrier easily• Hepatic metabolism
Dosage		<ul style="list-style-type: none">• Induction: 1 to 4.5 mg/kg IV• Sedation (IV): 1 to 2 mg/kg IV followed by 0.25 to 0.5 mg/kg IV every 5 to 10 min• Sedation (IM): 2 to 5 mg/kg IM
Indications		<ul style="list-style-type: none">• Dissociative anesthetic with analgesic properties• Induction agent• Sedation
Effects	CNS	<ul style="list-style-type: none">• Increased CBF and ICP, myoclonus<ul style="list-style-type: none">– Contraindicated in head trauma• Emergence delirium (visual/auditory hallucinations and confusion)<ul style="list-style-type: none">– Can be mitigated by administration of a benzodiazepine prior to initiation of ketamine
	CV	Increased blood pressure, heart rate, cardiac output
	Respiratory	<ul style="list-style-type: none">• Ventilation is not depressed significantly• Increases tracheobronchial secretions<ul style="list-style-type: none">– Pretreatment with glycopyrrolate may decrease hypersalivation• Bronchodilation
NMDA, N-methyl-D-aspartate; IV, intravenously; IM, intramuscularly; CNS, central nervous system; CV, cardiovascular.		

Benzodiazepines

Examples		Midazolam, diazepam, lorazepam
Mechanism of action		GABA mediated
Dosage		See dosage chart on page 58
Effects	CNS	<ul style="list-style-type: none">• Sedation• Muscle relaxation• Retrograde amnesia• Anticonvulsant• Hypnotic• Decreased CBF
	CV	Mild vasodilation
	Respiratory	Only mild dose-dependent depression of ventilation

(Benzodiazepines cont)

Drug interactions	Erythromycin decreases metabolism of midazolam
Reversal agent	<ul style="list-style-type: none">• Flumazenil: Competitive antagonist<ul style="list-style-type: none">– Adult dose: 0.2 mg IV; repeated doses may be given at 1-minute intervals until a maximum of 3 mg– Pediatric dose: 0.01 mg/kg; may be repeated at 1-minute intervals to a maximum dose of 1 mg• Half-life of flumazenil is less than that of the benzodiazepines so re-sedation is likely to occur

GABA, gamma-aminobutyric acid; CNS, central nervous system; CV, cardiovascular.

Benzodiazepine dosages

	Dose	Duration
Midazolam	0.02 to 0.1 mg/kg IVP or 0.25 to 1.5 mcg/kg/min infusion; 0.5 mg/kg orally	15 to 20 min IV
Diazepam	2.5 to 10 mg IV/orally	15 to 30 min IV
Lorazepam	1 to 4 mg IVP/orally or 0.01 to 0.05mg/kg/hr infusion	60 to 120 min IV

IVP, intravenous push; IV, intravenously.

Dexmedetomidine

- Dose: 1 mcg/kg IV loading dose; 0.6 mcg/kg/hour intravenous (IV) maintenance
- Use: Sedation (short term in intubated patients)
- Mechanism of action: α_2 -adrenoceptor agonist
- Effects
 - Central nervous system (CNS): Rousable sedation, analgesia, anxiolysis
 - Cardiovascular (CV): Decreased heart rate, systemic vascular resistance leading to decreased cardiac output and blood pressure
 - Respiratory: Minimal

Neuromuscular Blockers

- Indications: Skeletal muscle relaxation during intubation, ventilation, or during treatment of laryngospasm refractory to positive pressure
- Two types
 - Depolarizing: eg, succinylcholine
 - Nondepolarizing: eg, cisatracurium
- Reversal agent: Neostigmine—Anticholinesterase (nondepolarizing agents only)
 - Mechanism of action: Inhibits acetyl cholinesterase, leading to a buildup of acetylcholine (ACh) within the neuromuscular junction (NMJ)
 - Dosage: 0.03 to 0.07 mg/kg over 1 minute
 - Side effect: Bradycardia (minimized with concurrent administration of atropine or glycopyrrolate)

Depolarizing agent	Succinylcholine
Mechanism of action	<ul style="list-style-type: none">• Competitive agonist at ACh receptor• Produces a sustained depolarization of the postjunctional membrane of the NMJ
Pharmacokinetics	Hydrolyzed by plasma cholinesterase
Onset	<ul style="list-style-type: none">• Occurs in 30 to 60 seconds• Associated with muscle fasciculations
Dosage	<ul style="list-style-type: none">• Adult induction: 0.6 mg/kg IV• Treatment of laryngospasm: 0.1 to 0.5 mg/kg IV or 4 to 6 mg/kg IM
Side effects	<ul style="list-style-type: none">• Fasciculations• Hyperkalemia• Cardiac dysrhythmias• Malignant hyperthermia
Contraindications and precautions	<ul style="list-style-type: none">• Recent burn/crush injuries (potential cardiac arrest/arrhythmias)• Neuromuscular injuries (prolonged effects)• Narrow angle glaucoma• Malignant hyperthermia• Skeletal muscle myopathies (can cause rhabdomyolysis; elevated potassium level)
IM, intramuscularly.	

Nondepolarizing Agents

Differences in onset, duration, and metabolism exist among the various options in this category.

Examples	Rocuronium, atracurium, cisatracurium, vecuronium, pancuronium
Mechanism of action	Competitive antagonists of ACh at postjunctional membrane
Pharmacokinetics	Cisatracurium: Degraded via Hoffman elimination (can be used in renal or hepatic failure patients)
Onset	<ul style="list-style-type: none">• Rocuronium: 75 seconds• Atracurium: 110 seconds• Cisatracurium: 150 seconds• Vecuronium: 180 seconds• Pancuronium: 220 seconds
Dosage	<ul style="list-style-type: none">• Rocuronium: 0.6 to 1.2 mg/kg• Atracurium: 0.4 to 0.5 mg/kg• Cisatracurium: 0.2 mg/kg• Vecuronium: 0.1 mg/kg• Pancuronium: 0.04 to 0.1 mg/kg

Local Anesthetics

Classification

- Amides: Metabolized by liver microsomal enzymes; incidence of allergic reaction is lower than with esters and likely related to the preservative rather than the anesthetic itself
- Esters: Metabolized by plasma pseudocholinesterase (consider if severe liver disease); *p*-aminobenzoic acid is a metabolite and may be associated with allergy in some patients
- Patients with sulfa allergies may have a higher incidence of allergy to esters

Mechanism of action

- Inhibits depolarization and impulse propagation of nerve cells primarily through blockade of sodium ion channels in the inactivated state

Pharmacokinetics

Property	Correlation	Description
pK_a	Onset	Decreased pK_a leads to faster onset
Lipid solubility	Potency	Increased solubility leads to increased potency
Protein binding	Duration	Increased binding leads to increased duration

Local anesthetic agents

	Duration of action		Maximum dosage
	Soft tissue	Pulp	
2% Lidocaine (1:100,000 epinephrine)	3–5 hr	60 min	4.4 mg/kg or 300 mg
2% Mepivacaine (1:200,000 levonor)	2–4 hr	60 min	6.6 mg/kg
4% Prilocaine (1:200,000 epinephrine)	3–8 hr	60–90 min	6 mg/kg or 400 mg
4% Articaine (1:100,000 epinephrine)	3–6 hr	60 min	7 mg/kg or 500 mg
0.5% Bupivacaine (1:200,000 epinephrine)	4–9 hr	90–180 min	1.3 mg/kg or 90 mg
3% Mepivacaine	90–120 min	20–40 min	6.6 mg/kg or 400 mg
4% Prilocaine	60–120 min (infiltration) 220–240 min (block)	10–15 min (infiltration) 40–60 min (block)	8 mg/kg or 600 mg

Systemic effects

CNS	<ul style="list-style-type: none">CNS depression and sedationAt high doses, inhibition of central inhibitory pathways may result in seizure activityIn the early stages of overdose, excitatory signs and symptoms may be observed (twitching, tremors, tinnitus)
CV	<ul style="list-style-type: none">Myocardial depression (rate, contractility, conduction) and peripheral vasodilation (exception—cocaine produces vasoconstriction)Of note, procaine causes the greatest vasodilation and is best for prevention of ischemia associated with inadvertent intra-arterial injections
Respiratory	<ul style="list-style-type: none">Bronchial smooth muscle relaxationRespiratory depression with overdose

Mechanical Ventilation

Two major types:

- Volume-controlled ventilation: Flow rate, respiratory rate, and tidal volumes are set, allowing for variable airway pressures
- Pressure-controlled ventilation: Inspiratory pressure and the inspiratory to expiratory (I:E) ratio are set while tidal volume is variable

Mode	Description
Continuous mandatory ventilation (CMV)	<ul style="list-style-type: none">Minute ventilation controlled by the ventilatorNo patient-initiated breathsVolume or pressure control can be selectedMost appropriate when patient can make no effort to breathe or when ventilation must be completely controlled<ul style="list-style-type: none">DrugsCerebral malfunctionsSpinal cord injuryPhrenic nerve injuryMotor nerve paralysis
Assist control ventilation	<ul style="list-style-type: none">Baseline minute ventilation set by the ventilatorPatient-initiated breaths trigger the ventilator to provide a controlled breath based on a set tidal volume or set pressure (depending on volume or pressure control)Indications<ul style="list-style-type: none">Patients requiring full ventilatory supportPatients with stable respiratory driveAdvantages<ul style="list-style-type: none">Decreases the work of breathingAllows patients to regulate respiratory rateHelps maintain a normal partial pressure of carbon dioxide (PaCO₂)Complications: Alveolar hyperventilation

(Mechanical Ventilation cont)

Synchronized intermittent mandatory ventilation (SIMV)	<ul style="list-style-type: none">• Baseline minute ventilation set by the ventilator• Patients can take additional spontaneous breaths that are not supported by the ventilator• Version of intermittent-mandatory ventilation in which the ventilator breaths are synchronized with the patient's efforts• Indications: Facilitate transition from full ventilatory support to partial support• Advantages<ul style="list-style-type: none">– Maintains respiratory muscle strength by avoiding muscle atrophy– Decreases mean airway pressure– Facilitates ventilator discontinuation, "weaning"• Complications<ul style="list-style-type: none">– When used for weaning, may be done too quickly and cause muscle fatigue– Mechanical rate and spontaneous rate may be asynchronous, causing "stacking" (incomplete expiration resulting in residual air adding to volume of next inspiration)– May cause barotrauma or volutrauma
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Spontaneous modes

Partial specific volume (PSV)	<ul style="list-style-type: none">• Pressure support given during inspiration until the inspiratory flow drops to a preset percentage of its peak flow• Breaths must be initiated by the patient; there is no set respiratory rate• Can be combined with SIMV so that pressure support is provided during the patient's spontaneous breaths; will decrease some of the work of breathing associated with the resistance of the circuit
Continuous positive airway pressure (CPAP)	<ul style="list-style-type: none">• Positive pressure provided continuously throughout inspiration and expiration• Breaths are initiated by the patient
Bilevel positive airway pressure (BiPAP)	<ul style="list-style-type: none">• Inspiratory and expiratory positive airway pressure is preset• The difference between the two pressures determines the tidal volume
Positive end-expiratory pressure (PEEP)	<ul style="list-style-type: none">• Positive pressure only applied during the expiratory phase• Helps prevent early airway closure and alveolar collapse at the end of expiration by increasing (and normalizing) the functional residual capacity (FRC) of the lungs

Extubation Criteria

Negative inspiratory force (NIF)	< -30 cm H ₂ O
Forced vital capacity (FVC)	> 10 cc/kg
Respiratory rate (RR)	< 35/min
Minute volume	< 10 Lpm
Tidal volume (TV)	> 5 cc/kg
Rapid shallow breathing index (RSBI) = f/TV	< 105
FiO ₂	< 60%
PaO ₂ /PaCO ₂	Stable
pH	7.35 to 7.45
Mental status	Alert/cooperative
PaO ₂ , partial pressure of oxygen.	

Perioperative Care

Cardiovascular Problems

Hypertension	<div>1. Determine the etiology<ul style="list-style-type: none">• Common causes: Drug errors, pain, preexisting essential hypertension, hypoxia• Uncommon causes: Malignant hyperthermia, pheochromocytoma, hyperthyroidism, fluid overload</div> <div>2. Confirm blood pressure</div> <div>3. Treat contributing factors (analgesics/deep anesthesia)</div> <div>4. Discontinue surgical stimulation</div> <div>5. Consider antihypertensive: Beta blockers (esmolol/labetalol), calcium channel blockers, hydralazine, nitroprusside, nitroglycerin</div> <div>Hypertension with bradycardia<ul style="list-style-type: none">• Hydralazine (2.5 to 5.0 mg IV every 10 minutes up to a maximum dose of 25 mg) or nitroglycerin sublingual tablet (0.4 mg every 5 minutes)</div> <div>Hypertension with tachycardia<ul style="list-style-type: none">• Esmolol (5 to 10 mg every 3 minutes up to a maximum dose of 100 to 300 mg) or labetalol (5 to 10 mg every 10 minutes up to a maximum dose of 300 mg)</div>
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(Cardiovascular Problems cont)

Hypotension	<ol style="list-style-type: none">1. Determine etiology: Hypovolemia, administered medications/depth of anesthesia, cardiogenic (dysrhythmia, myocardial infarction), pulmonary (pulmonary embolism, pneumothorax), anaphylaxis<ul style="list-style-type: none">• Hypertensive patients who are taking a diuretic and angiotensin-converting enzyme (ACE) inhibitor and/or angiotensin receptor blocker (ARB) are more prone to experience hypotension.2. Recheck blood pressure3. Intravenous fluid bolus should be the first line of treatment (250 mL of lactated Ringer [LR] or normal saline solution [NSS] or 10 mL/kg)4. Treat contributing factors: Adjust volatile agents, optimize preload <p>Hypotension with bradycardia</p> <ul style="list-style-type: none">• Atropine 0.5 mg every 3 to 5 minutes up to a maximum dose of 3.0 mg <p>Hypotension with tachycardia</p> <ul style="list-style-type: none">• Phenylephrine 1% (alpha agonist with reflex bradycardia effect) 100 µg per dose every 5 minutes <p>Hypotension with normal heart rate</p> <ul style="list-style-type: none">• Ephedrine (alpha and beta agonist) 5 mg every 5 to 10 minutes <p>Hypotension related with anaphylactic reaction</p> <ul style="list-style-type: none">• Epinephrine intramuscularly (IM) 0.3 mg of 1:1,000 (mild case); epinephrine IV 10 to 20 µg 1:10,000 (severe case)
Tachycardia	<ol style="list-style-type: none">1. Determine if sinus tachycardia; obtain ECG2. Common causes of sinus tachycardia perioperatively include pain, anxiety; other causes include fluid depletion, hypoxia, pulmonary embolism, residual effects of anesthetics/medications, hyperthyroidism, acute coronary syndrome, chronic obstructive pulmonary disease3. If tachyarrhythmia (vagal maneuvers, adenosine if paroxysmal supraventricular tachycardia), refer to advanced cardiovascular life support (ACLS) guidelines
Bradycardia	<ol style="list-style-type: none">1. Determine if symptomatic or unstable2. Commonly a result of residual drug effect if no preoperative history; consider reversal of causative medicine if symptomatic (eg, naloxone)3. If unstable: Atropine 0.5 mg every 3 to 5 minutes (up to 3 mg)4. Transcutaneous pacer and consider dopamine (2 to 10 mcg/kg/minute) or epinephrine (2 to 10 mcg/minute) infusion; refer to ACLS guidelines if persistent
Angina	<ol style="list-style-type: none">1. Stop procedure2. 100% oxygen3. Check vital signs4. Obtain ECG5. Administer aspirin (325-mg tablet chewed)6. If no hypotension, administer sublingual nitroglycerin, 0.3 to 0.6 mg every 5 minutes as needed, up to 3 doses in 15 minutes7. Morphine for ongoing pain8. Activate emergency medical services (EMS) or transfer if persistent pain or signs of ischemia on ECG9. Analyze cardiac enzymes (troponins) three times, 8 hours apart

(Cardiovascular Problems cont)

Syncope	<ol style="list-style-type: none">1. Reposition patient: Trendelenberg/supine2. Administer oxygen3. Check vital signs4. Rule out other nonsyncopal conditions resulting in loss of consciousness, such as acute coronary syndrome5. Consider additional testing: ECG, orthostatic challenge
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Respiratory Problems

Laryngospasm	<p>Findings: Little to no ventilation, inspiratory stridor, paradoxical chest/abdominal movements</p> <ol style="list-style-type: none">1. Suction airway and pack the surgical site2. Administer 100% oxygen3. Use positive pressure ventilation (bag and mask)4. Consider deepening the anesthesia5. Consider succinylcholine (rocuronium, if contraindicated)
Bronchospasm	<p>Findings: Expiratory wheezing, prolonged expiration, increased peak airway pressure</p> <ol style="list-style-type: none">1. Rule out allergic reaction, aspiration2. Administer 100% oxygen3. Use albuterol inhaler4. Epinephrine, if refractory5. Consider intubation; if already intubated, consider deepening the level of anesthesia or heliox therapy (gas composed of helium and oxygen)

Malignant Hyperthermia

- Hypercatabolic state that develops in genetically susceptible individuals as a response to a variety of in-halational anesthetics (sevoflurane, desflurane, isoflurane) as well as succinylcholine

Pathogenesis	Mediated by calcium release from the sarcoplasmic reticulum in skeletal muscle
Presentation	<ul style="list-style-type: none">• Early signs: hypercapnia, tachycardia, muscle rigidity• Later signs: ECG changes secondary to hyperkalemia, rhabdomyolysis (elevated plasma creatine kinase and urine myoglobin—dark urine), and hyperthermia.<ul style="list-style-type: none">– Most common causes of death are hyperkalemia and coagulopathy from hyperthermia
Treatment	<ol style="list-style-type: none">1. Stop triggering agent2. Administer dantrolene: 2.5 mg/kg IV loading bolus followed by additional 1 mg/kg IV boluses until symptoms have resolved3. Continue supportive care, oxygen, cooling blankets, place Foley catheter4. Correct electrolytes (hyperkalemia treatment includes glucose/insulin, calcium gluconate, and furosemide)

Fluid Management

Maintenance fluid requirements	<ul style="list-style-type: none">• First 10 kg: 4 cc/kg/hour• Second 10 kg: 2 cc/kg/hour• Each additional 10 kg: 1 cc/kg/hour• Nils per os (NPO) deficit = number of hours NPO \times maintenance requirement• Free water deficit: $0.6 \times \text{weight (kg)} \times \left(\frac{\text{serum sodium}}{140} - 1 \right)$• Crystalloid: Sterile water with added electrolytes (normal saline, LR solution)• Colloid: Contains large molecular weight substances with less membrane permeability to allow for increased intravascular osmotic pressure (albumin, hetastarch)
Red blood cells (RBCs)	<ul style="list-style-type: none">• Allowable blood loss<ul style="list-style-type: none">– $[\text{estimated blood volume} \times (\text{hematocrit} - \text{target hematocrit})] \div \text{hematocrit}$– Each unit of RBCs increases hematocrit by 3% and hemoglobin by 1%• To restore volume after blood loss<ul style="list-style-type: none">– 3 cc of crystalloid are used for every 1 cc of blood lost– If using RBCs or colloid to restore volume, a 1:1 ratio is used• Risks of transfusion include<ul style="list-style-type: none">– Hemolytic/nonhemolytic immune reactions– Infection (viral/bacterial/parasitic)– Coagulopathy disorders (eg, disseminated intravascular coagulation)• Administer product using normal saline (avoid using LR solution)
Platelets	<ul style="list-style-type: none">• 1 unit of platelets will increase the count by 5,000 to 10,000/mm³ (normal count is 150,000 to 440,000/mm³)• In general, a count of at least 50,000/mm³ is required for surgery, although the type of surgery and any preexisting bleeding diatheses may change the threshold
Fresh frozen plasma (FFP)	Contains all of the coagulation factors; may be used for reversal of warfarin as well as replacement of deficient factors
Cryoprecipitate	Enriched with fibrinogen as well as von Willebrand factor, factor VIII-C, and factor XII

Postoperative Management

- Various criteria must be evaluated to ensure safety prior to discharge
- Most protocols involve assessment of the following: Consciousness, breathing, oxygen saturation, circulation, activity

Modified Aldrete discharge criteria

- Maximum score is 10
- Patient should not be discharged when score is < 8

	2	1	0
Respiration	Normal depth and rate of respiration	Dyspnea/shallow breathing	Airway support needed
Oxygen saturation	> 92%	Needs oxygen to maintain 90%	< 90% with oxygen
Consciousness	Alert and oriented ×3	Arousable by verbal command	Arousable by physical stimulus
Circulation	BP +/- 20 mm Hg of anesthetic level	BP +/- 20 to 50 mm Hg of anesthetic level	BP outside +/- 50 mm Hg of anesthetic level
Activity	Moves extremities voluntarily or on command	Moves two extremities	No extremity movement
BP, blood pressure.			

Common postoperative issues

Nausea and vomiting	<ul style="list-style-type: none"> • Multiple receptors converge at the chemoreceptor trigger zone • Targets for antiemetic therapy include – <ul style="list-style-type: none"> – Serotonin antagonism (ondansetron, dolasetron) – Histamine: Promethazine (primarily H1 receptor, also acetylcholine receptor, 5-hydroxytryptamine receptor, dopamine receptor [D2]) – Dopamine: Droperidol (concerns for QT wave prolongation), metoclopramide (primarily D2, has prokinetic properties as well) – Muscarine receptor: Scopolamine (can be applied transdermally) – Corticosteroids such as dexamethasone can also be used; commonly given at the beginning of the surgery
Delayed awakening	<ul style="list-style-type: none"> • Causes: Overdose, hypothermia, hypercarbia, hypoxia, cerebrovascular accident • Pre-hospital stroke assessment includes exam of speech, facial weakness, and arm drift in addition to routine vital signs/labs and supportive care • Evaluate vital signs and temperature in addition to electrolytes and glucose level • Consider the following <ul style="list-style-type: none"> – Naloxone to reverse opiates – Flumazenil to reverse benzodiazepines – Physostigmine for reversal of anticholinergics
Airway compromise and hypoventilation	<ul style="list-style-type: none"> • Usually a result of residual effects from anesthesia • Administer oxygen and consider reversal of any medications • With upper airway obstruction, laryngospasm must be considered; treatment involves oxygen via positive pressure ventilation/jaw thrust and administration of a paralytic (succinylcholine)
Agitation and pain	Consider using nonnarcotic agents when possible

Emergency medicines administered via endotracheal tube

- Volume: For adults, dilute the volume to 10 mL; for children dilute to a volume of 5 mL
- Dose:
 - Adult: 2 to 2.5 × IV dose
 - Child: Variable
- Follow dose with five ventilations
 1. Naloxone: 2 to 2.5 × standard dose
 2. Atropine: 2 to 2.5 mg (child: 0.04 to 0.6 mg/kg)
 3. Vasopressin: 40 units + 10 cc normal saline
 4. Epinephrine: 2 to 2.5 mg (child: 0.1 mg/kg)
 5. Lidocaine: 2 to 4 mg/kg

Anesthesia for Special Populations

Pediatric Patients

Airway/ respiratory	Anatomic differences <ul style="list-style-type: none"> • Tongue relatively larger • Epiglottis is floppy (omega shaped) and located higher • Larynx is funnel shaped • Narrowest point of the airway is lower in the subglottic region Physiologic differences <ul style="list-style-type: none"> • Upper airway is more compliant and prone to compression from negative inspiratory forces • More reliant on diaphragm; accessory muscles may not contribute as much as in adults Intubation <ul style="list-style-type: none"> • Endotracheal (ET) tube selection (age > 2 years): $\text{Age} \div 4 + 4 = \text{mm of diameter for ET tube}$
Cardiovascular	Cardiac output is driven mostly by heart rate
Temperature	Larger surface: Volume ratio and decreased fat insulation make pediatric patients more prone to drops in body temperature; keep room temperature higher than for an adult patient

Elderly Patients

- In the absence of comorbidities, age is not an independent risk factor for anesthetic complications
- Global decline in organ function leads to less reserve

CNS	<ul style="list-style-type: none"> • Decreased minimal alveolar anesthetic concentration (increased sensitivity to volatile agents) • Increased incidence of postoperative delirium
Cardiovascular	<ul style="list-style-type: none"> • Hypertension (decreased vessel compliance), decreased cardiac output and heart rate
Respiratory	<ul style="list-style-type: none"> • Increased ventilation/perfusion (V/Q) mismatch
GI	<ul style="list-style-type: none"> • Decreased gastric motility/emptying, decreased lower esophageal sphincter tone leads to aspiration
Pharmacokinetics	<ul style="list-style-type: none"> • Decreased clearance of drugs results from decreased renal clearance and hepatic metabolism • Decreased total body water and an increased body fat/muscle ratio results in differential effects on the volume of distribution, which is increased with fat-soluble medications but decreased with water-soluble medications in addition to those metabolized in muscle
CNS, central nervous system; GI, gastrointestinal.	

Obese Patients

- Body mass index (BMI) = kg/m^2
 - Overweight = BMI > 25
 - Obese = BMI > 30
 - Severe/morbid obesity = BMI > 40
- A careful airway examination is necessary as obesity may cause difficulty during intubation and mask ventilation
- Neck circumference
 - Single biggest predictor of problematic intubation in morbidly obese patients
 - 40-cm neck circumference = 5% probability of a problematic intubation
 - 60-cm neck circumference = 35% probability of a problematic intubation
- Sedation is challenging in these patients, especially if there is obstructive sleep apnea or Pickwickian syndrome (obesity hypoventilation syndrome)
- Aspiration risk precautions, especially if diabetic (gastroparesis)

(Obese Patients cont)

Cardiovascular	Poor cardiac compliance (caution about overload in fluid replacement)
Respiratory	Anatomical differences <ul style="list-style-type: none"> • Limitation of movement of the atlantoaxial joint and cervical spine • Excessive tissue folds in the mouth and pharynx • Short thick neck Physiologic differences <ul style="list-style-type: none"> • Decreased chest wall/lung compliance • Decreased functional residual capacity; adequate pre-oxygenation is necessary prior to intubation <ul style="list-style-type: none"> – Reverse Trendelenburg position – Increased compliance results in lower airway pressures
Pharmacokinetics	<ul style="list-style-type: none"> • Reduced total body water/blood volume • Increased body fat • Increased renal clearance because of increased glomerular filtration rate • Highly lipophilic drugs have an increased volume of distribution (longer time to eliminate) • Less lipophilic drugs show little or no change in volume of distribution with obesity

Diabetic Patients

- Higher morbidity and mortality than nondiabetic patients perioperatively

Perioperative risks	<ul style="list-style-type: none"> • Dehydration (osmotic diuresis with hyperglycemia) • Autonomic dysfunction (postural hypotension) • Gastroparesis/increased aspiration risk • Wound healing • Infection • Stiff joints (may have limited neck extension during intubation) • Diabetic ketoacidosis risk (type 1 DM) • Hypoglycemia
Management	<ul style="list-style-type: none"> • Schedule type 1 DM patients early morning if possible • Obtain glucose level on arrival • Consider halving the daily dose of long-acting insulin (depending on the severity and duration of surgery)

(Diabetic Patients cont)

Hyperglycemia	<ul style="list-style-type: none">• Generally asymptomatic in mild and moderate cases• Severe cases can present with diabetic ketoacidosis (type 1 DM) or hyperglycemic hyperosmolar nonketotic (HHNK) state (type 2 DM).• Symptoms<ul style="list-style-type: none">– Tachypneic, tachycardia, abdominal pain, temperature alteration– Ketone breathe in type 1 DM• Management – Activate EMS in office setting – Administer<ul style="list-style-type: none">◦ Regular insulin infusion (0.1 units/kg/h)◦ Normal saline 5 to 10 mg/kg/h (add 5% glucose when blood sugar < 250 mg/dL)◦ Replenish potassium (0.3 to 0.5 mEq/kg/h)
Hypoglycemia	<ul style="list-style-type: none">• Symptoms: Mental status change, diaphoresis, tachycardia, possible seizure disorder• Management – No change in mental status: Oral glucose-containing solution – Change in mental status<ul style="list-style-type: none">◦ No IV access: 1 to 2 mg IM glucagon◦ IV access: 10 to 25 gm glucose (ie, 20 to 50 mL 50% solution or 40 to 100 mL of 25% solution)
DM, diabetes mellitus.	

Hyperthyroid Patients

- Hyperthyroid (thyroid) crisis occurs in patients with underlying thyroid disease (eg, Graves disease, toxic multinodular goiter), resulting in hyperpyrexia, tachycardia, heart failure, CNS changes, GI disturbances

Precipitants	<ul style="list-style-type: none">• Infection, trauma, surgical stress, withdrawal/medicine noncompliance
Prevention	<ul style="list-style-type: none">• Ensure euthyroid state prior to elective surgery (thionamides)
Treatment	<ul style="list-style-type: none">• Supportive measures: Oxygen/IV fluids, acetaminophen, thionamides (propylthiouracil/methimazole), propranolol, and hydrocortisone (both help to reduce peripheral conversion of T4 to T3)• Iodine therapy is delayed until after propylthiouracil/methimazole administration• Correct electrolyte disturbances• Treat the underlying precipitant• Admission to intensive care unit
T4, thyroxine; T3, triiodothyronine.	

Neurologic Trauma Patients

- Care with cervical spine (cervical collar may not be removed; therefore sandbags are required around the head to limit movement)
- Consider IV anesthetics instead of inhalational anesthetics to limit vasodilation in the setting of high intracranial pressure
- Fluid resuscitation with isotonic fluids only (normal saline)
- Keep head elevated
- Avoid ketamine

Myasthenia Gravis Patients

- Minimize dose of ester anesthetics (especially if patient receiving anticholinesterase therapy)
- Patients on anticholinesterase therapy will have decreased metabolism of succinylcholine
- Consider stress dose of steroid if on chronic steroid therapy
- Minimize premedications that may cause respiratory depression given low respiratory reserve
- Ensure sustained respiratory muscle strength prior to extubation
- Both myasthenic crisis (insufficient medication) and cholinergic crisis (excess medication) may present similarly: Muscle weakness, bronchospasm, wheezing, respiratory failure, diaphoresis, and cyanosis
- Treatment of myasthenia gravis includes: Anticholinesterases, thymectomy, immunosuppression, plasma exchange, and IV immunoglobulin

Asthmatic Patients

- Laryngeal mask rather than ET tube should be considered whenever possible
- IV induction agents: Propofol and ketamine both inhibit bronchoconstriction
- Volatile agents: Enflurane and isoflurane are potent bronchodilators (may be used in status asthmaticus)
- Paralytics: Vecuronium, rocuronium, cisatracurium, and pancuronium do not induce bronchospasm, whereas atracurium and mivacurium should not be used because they may cause histamine release
- Beta 2 agonists are the mainstay of treatment of acute bronchoconstriction
- Steroids are helpful for treatment of persistent asthma; with acute exacerbations, steroid may take up to 6 hours for effect

Patients with Cerebral Palsy

Definition	Cerebral palsy (CP) is a nonprogressive movement disorder that results from injury to the developing brain
Classification	<p>Based on motor function and deformity distribution</p> <p>Spastic CP</p> <ul style="list-style-type: none">• 75% of all CP (injury to the cerebral motor cortex)• Spastic diplegia (affects mostly lower extremities)• Spastic hemiplegia (ipsilateral extremities)• Spastic quadriplegia (all extremities); greater risk of epilepsy <p>Dyskinetic CP</p> <ul style="list-style-type: none">• Injury to the basal ganglia• Characterized by dystonia, athetosis, and chorea• Associated with impaired speech and drooling• 25% of dyskinetic CP patients have epilepsy <p>Ataxic CP</p> <ul style="list-style-type: none">• Injury to the cerebellum• Characterized by tremor, loss of balance, and difficulty with speech
Preoperative	<ul style="list-style-type: none">• Evaluate the degree of spasticity and limb contracture<ul style="list-style-type: none">– Can interfere with airway management, IV access, and surgical positioning• Evaluate history of seizure episodes• Evaluate severity of gastrointestinal reflux and impaired pharyngeal function with pooling of oral secretions• Evaluate respiratory function<ul style="list-style-type: none">– Reactive airway is a common finding in these patients<ul style="list-style-type: none">◦ History of recurrent pneumonia◦ Decreased airway tone– Presence of scoliosis and restrictive pulmonary disease
Intraoperative	<ul style="list-style-type: none">• Extremity contractures can make IV access difficult• Aspiration precautions• Volatile gas induction may be preferable route<ul style="list-style-type: none">– CP patient may need higher anesthetic concentration due to reduced minimal alveolar anesthetic concentration• Avoid succinylcholine due to hyperkalemic response• When using nondepolarizing skeletal muscle relaxants, CP patients may need higher dosage due to drug resistance• CP patient is also at risk for<ul style="list-style-type: none">– Pressure ulcer from prolonged positioning– Hypovolemia and pre-renal kidney failure

Recommended Readings

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Dentoalveolar Surgery

Esther S. Oh and George Blakey

- ▶ Management of Impacted Teeth Other Than Third Molars
- ▶ Management of Impacted Third Molars
- ▶ Soft Tissue and Preprosthetic Procedures
- ▶ Odontogenic Infections
- ▶ Associated Major Infections
- ▶ Temporary Anchorage Devices (TADs)
- ▶ Apicoectomy
- ▶ Miscellaneous Information

Management of Impacted Teeth Other Than Third Molars

Tooth Impaction

- Impacted tooth: Failure of a tooth to fully erupt in the oral cavity in the expected developmental period
 - Not all unerupted teeth are impacted
 - Not the same as ankylosed/submerged tooth
- Order of impaction frequency is reverse of eruption order: Third molars, maxillary canines, mandibular premolars, maxillary premolars, second molars (Fig 3-1)
- Rarely impacted: Mandibular incisors and first molars as well as primary teeth (versus ankylosis and submersion)

Tooth eruption sequence

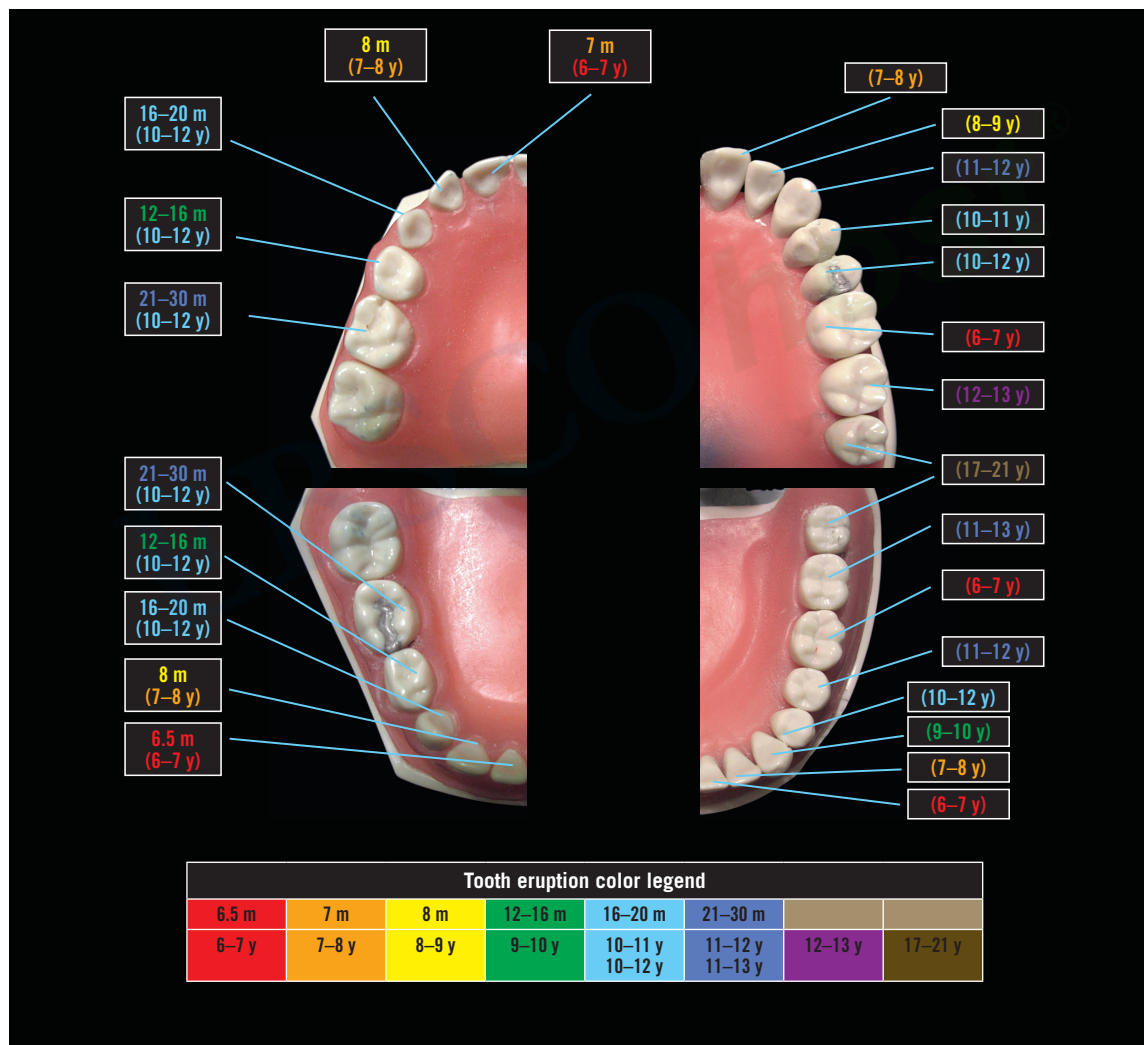


Fig 3-1 Tooth eruption chart showing eruption age and shedding age (in parentheses).

Factors associated with tooth impaction

Local factors	Systemic factors (usually multiple teeth)
<ul style="list-style-type: none">• Prolonged retention of primary tooth• Cleft lip/palate• Odontogenic cysts or tumors• Deficient arch length• Malposed tooth germ• Abnormal eruption path• Supernumerary tooth blockade	<ul style="list-style-type: none">• Cleidocranial dysplasia• Endocrine deficiencies (eg, hypothyroidism, hypopituitarism)• Febrile disease• Down syndrome• Irradiation

Determining location

- Clinical: Palpate, examine for bulge
- Radiographic options – Periapical (PA): “SLOB” or “shift” rule—same lingual (palatal), opposite buccal
 - After first PA, keep film position constant but move x-ray head either anterior or posterior
 - If impacted tooth appears to move in same direction as x-ray head, tooth is lingual (palatal)
 - If impacted tooth appears to move opposite to the x-ray head, the tooth is buccal
- Panoramic: If horizontal and/or possibly larger/out of focus, likely palatal; if vertical, likely buccal
- Cone beam computed tomography (CBCT): Primary modality or when standard radiographs provide equivocal results

Cone beam computed tomography

- Conventional medical CT scanner uses fan-shaped beam to obtain individual image slices, with each slice requiring a separate scan; slices stacked to get three-dimensional representation
- CBCT uses cone-shaped beam directed through middle of area and covers entire field of view; only one rotation needed, so less radiation
- Dose varies depending on system used and imaging protocols (slice thickness, field of view [FOV], mAs, kVp, scan time)
- ALARA radiation principle: “As low as reasonably achievable”

Radiation dose

- Gray (Gy) or rad: Absorption of 1 joule of radiation energy by 1 kg of matter
- Sievert (Sv): Effective dose or quantification of potential radiobiologic detriment (ie, cancer induction, genetic damage) from radiation
 - 1 Sv = 1,000 mSv = 1,000,000 μ Sv
 - Whole-body radiation dose of 1 Sv = estimated 4% to 5% increased relative risk of fatal cancer
 - 10,000 μ Sv single exposure = 1:1,000 persons exposed will develop cancer

Exposure type	μ Sv
Radiation from natural background in one year	3,000
Transcontinental flight	20
Single intraoral PA or bitewing radiograph	5
PA or lateral cephalometric radiograph	5.5
Panoramic radiograph	3–24
Full-mouth series radiograph	35
Small/medium FOV dentoalveolar CBCT	11–674*
Large FOV maxillofacial CBCT	30–1,073*
Single chest radiograph	20
CT of head	2,000
CT of neck	4,000
CT of chest	8,000
CT pulmonary embolism protocol	15,000
*Average radiation dose for CBCT is 61 for small/medium FOV and 87 for large FOV.	

Frequency, etiology, and management

	Impacted second molar	Impacted canine/premolar
Frequency	Maxillary canines > mandibular premolars > mandibular canines > second molars	
Etiology	Impacted third molar: Usually impacted second molar will erupt normally if obstructing third molar removed early enough	<ul style="list-style-type: none">• If labial, likely from arch-length deficiency• If palatal, likely from extra space in maxilla—excess growth, agenesis/peg lateral incisor, or stimulated eruption of lateral incisor or first premolar
Management	<p>Extraction</p> <ul style="list-style-type: none">• Removal of second molar to allow third molar substitution may succeed in maxilla, but not likely in mandible• May need to extract second molar if malformed, severely tipped, or deeply impacted <p>Surgical uprighting</p> <ul style="list-style-type: none">• Ideal situation: $\frac{2}{3}$ root formation and incomplete apical closure<ul style="list-style-type: none">– If too early (less than $\frac{2}{3}$ root), second molar may move into incorrect position– If too late (complete root formation), likely pulpal necrosis/calcification; possible root fracture <p>Technical tips</p> <ul style="list-style-type: none">• Extract third molar to create space for posterior tipping of second molar only if necessary; improves stability of second molar• Need intact cortical plates for stability—prevents buccal or lingual tipping• Avoid damaging second molar CEJ• Do not tip > 90 degrees• Stabilize to first molar and splint in place if tooth unstable• Ensure tooth out of occlusion	<p>Expose and bond</p> <ul style="list-style-type: none">• Use brackets instead of circumdental wires: Decreases external root resorption, easier to place, minimizes tissue removal, maintains keratinized tissue around tooth• Remove only enough soft tissue and bone to place bonded orthodontic bracket<ul style="list-style-type: none">– Do not expose CEJ—increased risk of root resorption, ankylosis, periodontal inflammation– If large palatal flap is needed, use palatal splint postoperatively to minimize dead space– Tip: As long as enough space is in the arch, both expose only and expose and bond have similar success, but most orthodontists prefer expose and bond– If mandibular premolar located in middle of alveolar process, bond bracket to occlusal surface– Apply vertical orthodontic forces until buccal surface exposed for new bracket placement <p>Soft tissue management</p> <ul style="list-style-type: none">• Apical mucoperiosteal flap with parallel mesial and distal releasing incisions<ul style="list-style-type: none">– Labially positioned impacted canine– Inadequate keratinized tissue<ul style="list-style-type: none">◦ Allows eruption into attached mucosa (avoid techniques that remove attached gingiva)◦ Less chance of periodontal defect and pocket formation

CEJ, cementoenamel junction.

Management of Impacted Third Molars

Normal Development and Eruption of Mandibular Third Molars (Fig 3-2)

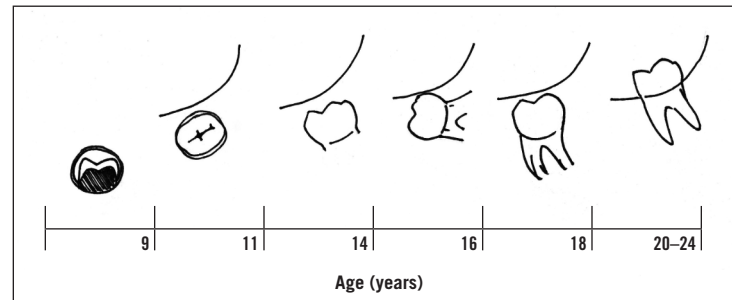


Fig 3-2 Development/eruption pattern of mandibular third molars (illustrated by Quoc Lu).

- Age 9: Radiographic evidence of tooth germ
- Age 11: Tooth germ at occlusal plane within anterior mandibular ramus, cusps mineralized, occlusal surface faces anterior
- Age 14: Crown formed
- Age 16: Roots 50% formed and at second molar root level with horizontal angulation; mandibular body grows in length as anterior ramus resorbs
- Age 18: Roots completely formed with open apices; as roots form, occlusal surface changes from anterior horizontal angulation to mesioangular to vertical position
- Age 20 to 24: Final tooth position with eruption completed

Possible Reasons for Third Molar Impaction

- 1. Different growth rates between mesial and distal roots with under- or over-rotation**
 - Underdeveloped mesial root with under-rotation = mesioangular impaction
 - Overdeveloped mesial root with over-rotation = distoangular impaction
 - Overdeveloped distal root (especially with mesial curve) = severe mesioangular or horizontal impaction
- 2. Relationship of bony arch length to sum of mesiodistal widths of teeth in arch**
 - Will have more impacted teeth with shorter arch or larger teeth
 - Abnormal lateral tooth position = impaction due to dense external oblique ridge
- 3. Retarded maturation. Tooth development lags behind skeletal growth/jaw maturation**
 - Tooth has decreased influence on mandible growth pattern/resorption
- 4. Attrition theory**
 - Refined diet so no mesial drift of remaining dentition

Tip: By age 18 to 20 years, if improper angulation or inadequate space, tooth will likely remain impacted

Impacted Third Molar Classification Systems

Pell and Gregory classification

Based on relation to occlusal plane and anterior border of ascending mandibular ramus.

- Maxillary and mandibular third molars based on relationship to occlusal plane (Fig 3-3)
 - Class A: Third molar occlusal plane is the same as the second molar occlusal plane
 - Class B: Third molar occlusal plane is between the second molar occlusal plane and cervical line
 - Class C: Third molar occlusal plane is below the second molar cervical line



Fig 3-3 Pell and Gregory classification of third molars based on relationship to occlusal plane. (a) Class A. (b) Class B. (c) Class C.

- Mandibular third molars based on relationship to anterior border of ramus (Fig 3-4)
 - Class I: Third molar has enough room to erupt anterior to the anterior border of the ramus
 - Class II: Half of third molar crown covered by ramus
 - Class III: Third molar is completely embedded in ramus



Fig 3-4 Pell and Gregory classification of mandibular third molars based on relationship to anterior border of ramus (highlighted in red). (a) Class I. (b) Class II. (c) Class III.

Winter classification

Based on radiographic anatomical position of third molar related to long axis of second molar (Fig 3-5).

- Mesioangular, 45%
- Distoangular, 5%
- Vertical, 40%
- Horizontal, 10%
- Linguoangular, rare
- Buccoangular, rare
- Inverted, rare

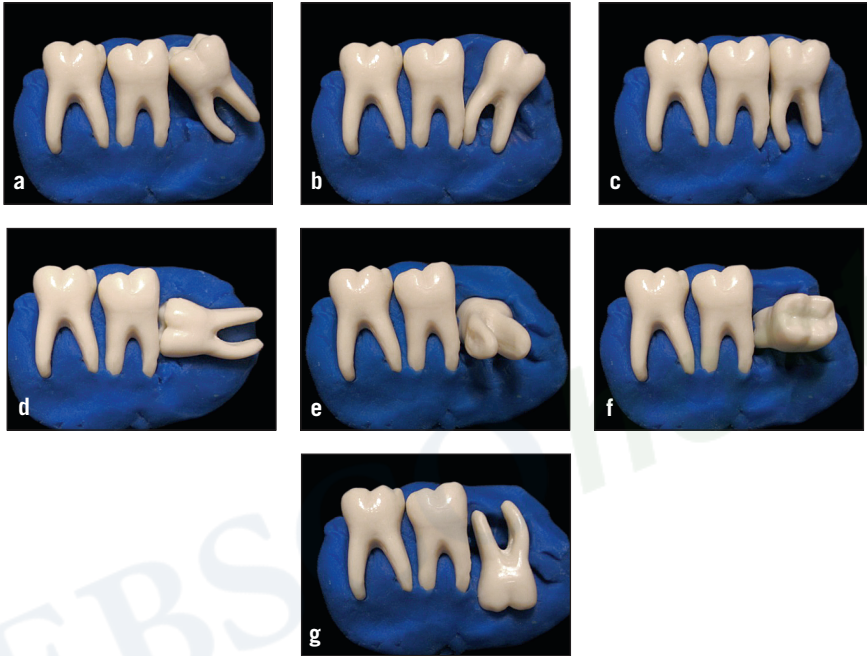
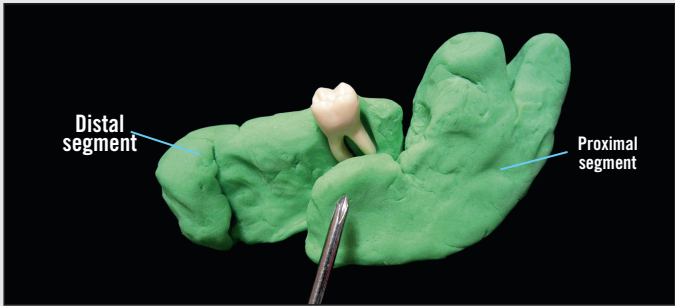


Fig 3-5 Winter classification of third molars. (a) Mesioangular. (b) Distoangular. (c) Vertical. (d) Horizontal. (e) Linguoangular. (f) Buccoangular. (g) Inverted.

ADA Codes for Impacted Third Molars with AAOMS Description

07220 (soft tissue impaction)	Removal requires incision of overlying soft tissue
07230 (partial bony impaction)	Requires incision of overlying soft tissue, flap elevation, and either bone/tooth removal or tooth sectioning
07240 (complete bony impaction)	Requires incision of overlying soft tissue, flap elevation, bone removal, and tooth sectioning
07250 (complete bony impaction with unusual surgical complications)	Requires incision of overlying soft tissue, flap elevation, bone removal, and tooth sectioning and involves an out-of-the-ordinary difficult situation
ADA, American Dental Association; AAOMS, American Association of Oral and Maxillofacial Surgeons.	

Indications for Third Molar Extraction

Pericoronitis	<ul style="list-style-type: none">• Most common indication for extraction after age 20• Often associated with operculum (soft tissue flap on distal of crown)• Acute inflammation with clinical signs and symptoms (pain, swelling, erythema, purulence)• Associated bacterial types found in gingival crevice of third molars and adjacent teeth<ul style="list-style-type: none">– Obligate and facultative anaerobes lead to purulence (<i>Peptostreptococcus</i>, <i>Fusobacterium</i>, and <i>Porphyromonas</i>) <p>Stages</p> <ul style="list-style-type: none">• Stage I: Inflammation only in pericoronal area• Stage II: Inflammation has spread to contiguous submucosa• Stage III: Inflammation has spread to adjacent spaces/fascial planes and stimulates systemic response <p>Treatment</p> <ul style="list-style-type: none">• Local measures: Irrigation (hydrogen peroxide or chlorhexidine), improve oral hygiene• +/- Antibiotics• +/- Extract offending opposing third molar (reduces trauma on operculum and helps relieve symptoms)• Operculectomy does not resolve problem; usually need to extract the tooth
Orthodontic problems	<p>Main issues</p> <ul style="list-style-type: none">• Cause of mandibular incisor crowding<ul style="list-style-type: none">– Not proven that third molars are responsible– Etiology is multifactorial and associated mainly with deficient arch length– No study able to isolate effect of third molars from all other factors causing crowding• Obstruction of orthodontic movement: May block distal orthodontic movement• Interference with orthognathic surgery – Advise removal of third molars 6 to 12 months preoperatively to allow adequate bone fill – With mandibular advancements, extraction during surgery reduces thickness/quality of lingual bone at proximal portion of distal segment (site of screw fixation) (Fig 3-6)<ul style="list-style-type: none">– During maxillary downfractures, deeply impacted third molars may be easier to extract through the maxillary sinus without vascular pedicle compromise <div data-bbox="657 1269 1334 1573"></div> <p>Fig 3-6 Third molars in orthognathic surgery.</p>

(Indications for Third Molar Extraction cont)

Periodontal disease	<p>If age < 25 years</p> <ul style="list-style-type: none"> • May use panoramic radiograph to assess adequacy of space between anterior border of mandibular ramus and distal of mandibular second molar for third molar eruption to occlusal plane • Impacted third molars may still change position after age 25 • Asymptomatic does not imply soft tissue health; especially horizontal and mesioangular impacted and visible third molars may have signs of periodontitis <ul style="list-style-type: none"> – Disrupted periodontal ligament – Root resorption – Pocket depths > 5 mm on distal surface of adjacent second molar – Attachment loss > 1 mm on distal surface of adjacent second molar – Second and third molar areas can have pathogenic “red” and “orange” microbial complexes (similar to microbes found in refractory periodontitis) – Third molar gingival crevicular fluid may have inflammatory mediators associated with systemic health risks <ul style="list-style-type: none"> ◦ Cardiovascular disease ◦ Nonhemorrhagic stroke ◦ Preterm low-birth weight pregnancy ◦ Kidney disease • Periodontal healing is best when third molar is extracted <ul style="list-style-type: none"> – Before exposure in mouth – Before bone resorption on distal of second molar • Gernectomy: Perform when < ½ root formation and radiographically discernible periodontal ligament <ul style="list-style-type: none"> – When patient is young with better healing capacity – Scaling/root planing and plaque control of adjacent second molar may help reduce post operative attachment loss <p>If age > 25 years</p> <ul style="list-style-type: none"> • Should not have asymptomatic third molars extracted unless pathology is present • More asymptomatic periodontal defects present (based on pocket depth) and higher caries prevalence • Third molar extraction may negatively affect adjacent second molar periodontium (30% risk of increased pocket depth) <ul style="list-style-type: none"> – Risk factors: Preoperative intrabony defect, age > 25 years old, poor plaque control • After age 19 years, second molar distal attachment levels and pocket depths are the same postoperatively as preoperatively • Slower recovery with higher economic hardship/time off work • Postoperative morbidity is 1.5 times greater
Associated cysts/tumors	<p>Curran et al study (see the recommended readings)</p> <ul style="list-style-type: none"> • Among patients > 50 years, 50% of 2,646 tissue specimens and 33% of study sample had a cyst/tumor <ul style="list-style-type: none"> – Dentigerous cyst (28%) > keratocystic odontogenic tumor (3%) > odontoma (0.7%) > ameloblastoma (0.5%) > carcinoma (0.23%) = calcifying odontogenic cyst (0.23%) > myxoma (0.04%) • Incidence of neoplastic changes in third molar follicle = 3%; usually seen in patients aged < 40 years, so possible decreased risk with increased age
Dental caries	<ul style="list-style-type: none"> • Usually in mandibular second or third molars, mostly at cervical line • Lack of space—unable to clean effectively • Dentist unable to access/isolate third molar effectively for restoration

(Indications for Third Molar Extraction cont)

Resorption of adjacent second molar root	<ul style="list-style-type: none"> • Incidence: Unclear but possibly up to 7% • Usually adjacent tooth repairs itself (deposits cementum and forms secondary dentin) <ul style="list-style-type: none"> – If resorption noted, extract third molar as soon as possible – If severe, may need to extract both teeth
Plan for overlying prosthesis	<ul style="list-style-type: none"> • Unerupted teeth are unpredictable (may still migrate), so ultimate treatment decision should be based on <ul style="list-style-type: none"> – Patient age – – Tooth position – – Type of overlying prosthesis – • Surgical risk: If tissue-borne prosthesis over impacted tooth only covered with soft tissue or 1 to 2 mm of bone, likely needs extraction; otherwise, bone resorption, mucosal perforation, pain/inflammation
Mandible fracture prevention	<ul style="list-style-type: none"> • Third molar presence increases risk of angle fracture 2.8-fold <ul style="list-style-type: none"> – Special consideration for athletes (contact sports) – Possibility of complications during fracture treatment if third molar in fracture line
Discomfort	<ul style="list-style-type: none"> • Discomfort from food impaction • Possible relief of unexplained pain <ul style="list-style-type: none"> – If no clinical/radiographic signs of pathology and all other sources ruled out, extraction of third molar may relieve pain – Inform patient prior to procedure that extraction may not relieve pain

Contraindications to Impacted Third Molar Extraction

Factors associated with increased complication incidence and severity

Patient factors	<p>Extremes of age</p> <ul style="list-style-type: none"> • Too young – May remove tooth bud by 8 to 9 years with minimal morbidity, but at that time unable to predict if tooth may eventually erupt into normal position • Too old (> 40 years) <ul style="list-style-type: none"> – Decreased healing capacity, so longer recovery and greater residual bony defect – – More dense, less flexible bone, so higher chance of fracture – – If complete bone coverage, no oral communication, and no pathology, extraction not usually recommended; however, will need long-term follow-up <p>Other factors</p> <ul style="list-style-type: none"> • Limited mouth opening, dense bone, lack of patient cooperation, ethnicity
Medical compromise	May need coordination with patient's physician for management
Anatomical factors	<ul style="list-style-type: none"> • Potential damage to nerves, adjacent teeth, and maxillary sinus, and other vital structures may be compromised • Tooth morphology, condition, depth in bone
Surgeon factors	Lack of experience

Complications Associated with Third Molar Extraction

Intraoperative complications

Root fracture	<ul style="list-style-type: none"> • Decreased incidence with adequate elevation of tooth before forceps delivery • Risk factors: Abnormal root curvature, dense bone, total root diameter > mesiodistal crown width • If root not infected and < 2 mm long, may leave without postoperative sequelae if removal would be complicated • If left in place, pulpal tissues become fibrous and root becomes totally incorporated within the bone <ul style="list-style-type: none"> – Need close radiographic follow-up – Must explain to patient and document thoroughly
Displaced tooth/roots	<p>Submandibular space</p> <ul style="list-style-type: none"> • Immediately apply upward external pressure in submandibular region medial to molars to prevent further displacement • If root seen through socket, retrieve with suction, root-tip pick, or grasp with small hemostat • If not seen, reflect subperiosteal lingual gingival flap extending anteriorly to canine; sharply detach mylohyoid muscle 4 mm from insertion to mandible; carefully dissect in region until visualize root • Prescribe postoperative antibiotics • If unable to retrieve root (location, uncontrollable bleeding, etc), wait 3 to 4 weeks to allow fibrosis/stabilization and then attempt as secondary procedure <p>Mandibular canal</p> <ul style="list-style-type: none"> • Verify location with periapical and occlusal radiographs • Rule out location in marrow space or under buccal mucosa • If clinically visible, carefully remove enough surrounding bone to grasp with fine hemostat/forcep • If not visible (location, brisk bleeding, etc), attempt as secondary procedure • Need to retrieve if persistent infection or paresthesia • If root asymptomatic, small, and not infected, may leave in place and inform patient <p>Maxillary sinus</p> <ul style="list-style-type: none"> • Verify location with periapical radiograph • Most common: Maxillary first molar palatal root • Buccal roots are usually displaced between buccal plate and periosteum • If between intact antral membrane and alveolar bone, slowly enlarge bony defect to find and remove • May have perforated sinus membrane, but root is still attached to alveolus by apical periodontal fibers • Local measures for retrieval – Place patient upright to prevent posterior root displacement – Close nostrils and have the patient blow through nose; root may appear at perforation – Fine-tip suction may bring root into extraction site – Antral lavage with sterile isotonic saline may flush root out through socket – If large opening, pack iodoform gauze into antrum and remove in one stroke; root tip may adhere to gauze <ul style="list-style-type: none"> – If local measures unsuccessful, use Caldwell-Luc (direct) approach in canine fossa – If profuse bleeding, likely from posterior superior alveolar artery – Postoperatively place figure-of-eight suture over extraction site; if large opening, may need to close with buccal or palatal flap – Initiate sinus precautions, antibiotics, and nasal spray; keep sinus ostium open, prevent infection

(Intraoperative complications cont)

Infratemporal space

- **Borders** (Fig 3-7)

- Superior/medial: Infratemporal crest of sphenoid bone
- Lateral: Temporalis muscle
- Inferior: Superior border of lateral pterygoid muscle
- Anterior: Posterior border of maxilla
- Posterior: Pterygomaxillary fissure and posterior border of orbit

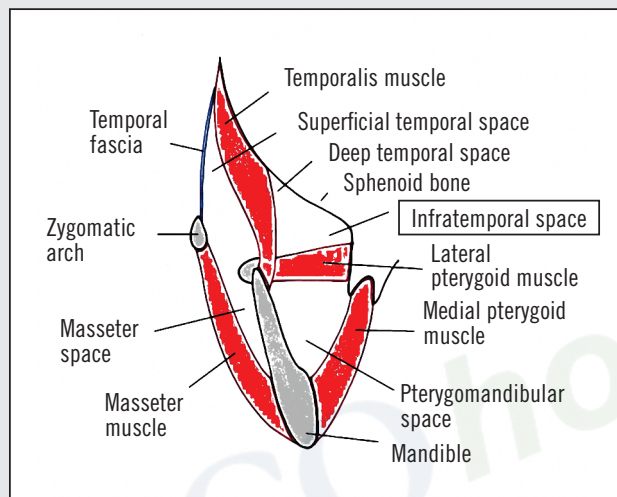


Fig 3-7 Borders of infratemporal space (illustrated by Quoc Lu).

- **Treatment**

- Place finger pressure high in buccal vestibule near pterygoid plates to try to manipulate tooth back into socket
- Use suction tip aimed posteriorly to try to guide tooth into socket
- If good access and light, make single cautious attempt to retrieve with hemostat
- If unsuccessful/unable to visualize tooth, close incision, inform patient, and prescribe antibiotics
 - After 3 to 4 weeks (to allow fibrosis), localize tooth using three-dimensional imaging
 - Under general and local anesthesia, use spinal needle to locate tooth
 - Dissect carefully along needle until tooth is visualized
 - Use right angle hemostat to get superior to tooth and guide it inferiorly
 - Irrigate, close wound, and prescribe postoperative antibiotics
- If no functional problems (tooth not impinging on zygomatic root causing trismus), and patient elects not to have tooth removed, properly document conversation

- **Prevention: Optimize visualization**

- Full-thickness mucoperiosteal flap
- Incision +/- releasing incision anterior to second molar
- Place broad retractor distal to third molar during elevation

(Intraoperative complications cont)

Aggressive bleeding	<p>Primary (intraoperative and immediate postoperative)</p> <ul style="list-style-type: none"> • Most common causes <ul style="list-style-type: none"> – Failure to suture – Torn tissue or surgical flaps – Granulation tissue: When pressure packs are removed, blood wells up into field <ul style="list-style-type: none"> ◦ Ensure adequate curettage and debridement to remove all granulation tissue – Periosteal blood vessels: Rebound blood vessel dilation <ul style="list-style-type: none"> ◦ Local anesthetic with vasoconstrictor may initially mask bleeding source ◦ Need good visualization, pressure packs, use cautery with caution – Bone nutrient vessels (especially anterior mandible) <ul style="list-style-type: none"> ◦ Burnish bone, crush surrounding bone, apply bone wax, use cautery with caution ◦ If severe bleeding, suspect concomitant nerve injury <p>Secondary (delayed postoperative)</p> <ul style="list-style-type: none"> • Identify source before giving local anesthetic with vasoconstrictor • Remove all sutures and remove clot from socket • Check bone and mucoperiosteal flap for active bleeding site • Do not use epinephrine-soaked packs (may have rebound vasodilation after vasoconstriction) • If simple generalized ooze, have patient place firm pressure on socket by biting on moist gauze pack for 5 minutes <p>Hemostatic agents</p> <ul style="list-style-type: none"> • Resorbable porcine skin gelatin sponge (Gelfoam, Pfizer) for capillary-type bleeding <ul style="list-style-type: none"> – Promotes platelet disruption then forms fibrin framework then blood clot – May be moistened with thrombin – Resorbs in 2 weeks • Oxidized cellulose plant polymer of polyanhydroglucuronic acid (Surgicel, Ethicon) <ul style="list-style-type: none"> – More efficient than gelatin sponge but more delayed healing – Forms matrix/scaffold then artificial blood clot formation – Resorbed between 7 and 14 days (depends on amount used, degradation rate, surrounding blood supply) – Note: When metabolized, creates acidic environment (pH = 2.8); may be reason for some reports of nerve dysfunction when placed in extraction sites • Type I bovine collagen plug (CollaPlug, Zimmer Dental) <ul style="list-style-type: none"> – Resorbed in 10 to 14 days • Thrombin: Bovine or recombinant human – Powder for reconstitution into aqueous solution – Activates blood coagulation factor IIa, then acts as serine protease to convert fibrinogen into fibrin • Antifibrinolytic topical mouthrinses <ul style="list-style-type: none"> – Aminocaproic acid: Inhibits plasmin – Tranexamic acid 5%: Inhibits conversion of plasminogen to plasmin <p>If severe and/or local measures unsuccessful</p> <ul style="list-style-type: none"> • Consider angiography for embolization • Consider patient/systemic factors <ul style="list-style-type: none"> – Arteriovenous malformation – Hemophilia A or B, von Willebrand disease – Anticoagulation medications • Consider coagulopathy work-up: complete blood count (CBC), prothrombin time (PT)/international normalized ratio (INR), partial thromboplastin time (PTT), bleeding time, factor 8 levels, ristocetin cofactor levels
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(Intraoperative complications cont)

Aspiration of foreign object	Foreign Object <ul style="list-style-type: none">Usually a tooth (especially maxillary third molar) Prevention <ul style="list-style-type: none">Gauze screen in oropharynx during all dentoalveolar procedures Treatment <ul style="list-style-type: none">Document details in patient chartIf non-acute (no airway problems)<ul style="list-style-type: none">Refer to emergency department for evaluationPosteroanterior/lateral chest/abdomen radiographs to determine presence in respiratory versus gastrointestinal tractIf respiratory tract, retrieve via bronchoscopyIf gastrointestinal tract, recommend bulky, high-roughage diet to help protect gastrointestinal tract<ul style="list-style-type: none">Instruct patient to check stool to confirm passage through gastrointestinal tract (about 3 days)If acute (airway obstruction/cyanosis), follow basic life support guidelines, possible surgical airway
Broken instrument (does not infer surgeon negligence)	<ul style="list-style-type: none">Stop procedure and suction without irrigation and localize fragmentMay migrate under mucoperiosteal flap into cancellous bone spaces, maxillary sinus, mandibular canal, or be suctioned awayIf not visualized, take radiograph that includes the entire surgical siteMake reasonable attempt to remove fragment at initial surgeryIf secondary procedure performed, need separate consentIf risks > benefits of removal, disclose/discuss with patient and document

Iatrogenic damage to adjacent teeth/structures

Hard tissue	<ul style="list-style-type: none">Tooth luxation: Stabilize 14 daysFracture restoration/crown: Contact dentistExtraction of wrong tooth: Contact dentist; if preorthodontic, contact orthodontist to see if treatment plan can be changedOsseous plate fracture: If attached to periosteum, consider leaving in place; if not, remove +/- graft as needed
Soft tissue	<ul style="list-style-type: none">Tear of mucosal flapThermal/rotary injury to lip from bur/handpiece

(Iatrogenic damage to adjacent teeth/structures cont)

Maxillary tuberosity fracture	<p>Risk factors</p> <ul style="list-style-type: none"> • Single, isolated maxillary molar • Bulbous, divergent, dilacerated roots • Use of excessive, uncontrolled force <p>Prevention</p> <ul style="list-style-type: none"> • If multiple teeth present, extract most distal tooth first • Expand buccal plate with elevator or osteotome • Section tooth <p>Treatment</p> <ul style="list-style-type: none"> • Stop and stabilize tooth with splint or wires and take tooth out of occlusion <ul style="list-style-type: none"> – Allow 4 to 6 weeks of healing before re-attempting extraction • If immediate tooth extraction indicated <ul style="list-style-type: none"> – Separate tooth from bone while stabilizing bone fragment – Section tooth to remove it – Suture overlying mucosa or use intraosseous wires to stabilize segment – If cannot separate tooth from bone, raise buccal subperiosteal flap and elevate tooth toward the buccal <ul style="list-style-type: none"> ◦ Elevate palatal mucosa from palatal plate ◦ Prevent mucosal tear, if possible, especially on palate • If no soft tissue left on avulsed tuberosity, remove it and smooth remaining bone edges • Check for oroantral communication and close wound
Oroantral communication	<p>Risk factors</p> <ul style="list-style-type: none"> • Incidence rate of 0.008% to 0.25% • First molar site • Large sinus • Acute/chronic sinusitis • Large periapical lesion/infection • Adjacent edentulous spaces • Traumatic extraction • Large, divergent roots <p>Opening size</p> <ul style="list-style-type: none"> • Bone defect larger than mucosal defect • If small opening suspected, do not enlarge, probe, irrigate, or have patient blow through occluded nostrils <ul style="list-style-type: none"> – May cause bigger perforation and/or infection • If opening < 2 mm, place suture across socket to support clot; caution patient not to blow nose for 1 week; no further treatment • If opening = 3 to 6 mm, gelatin sponge pack, figure-of-eight suture over socket, sinus precautions • If opening > 6 mm, tension-free primary closure; excise fistula and invert into sinus

(Iatrogenic damage to adjacent teeth/structures cont)

	<p>Options for closure</p> <ul style="list-style-type: none"> • Buccal advancement or buccal finger flap • Palatal island or finger flap • Pedicled buccal fat pad: Heals by secondary epithelialization <ul style="list-style-type: none"> – Blood supply: Internal maxillary artery branches • Tongue flap: Make flap 5 to 7 mm thick and 20% wider than defect – Anterior-based blood supply, deep lingual artery: For defects in anterior alveolar ridge and hard palate <ul style="list-style-type: none"> – Posterior-based blood supply, suprahyoid artery and dorsalis lingual artery: For defects in posterior alveolar ridge and soft palate – Can rotate half tongue without compromising normal function – Need maxillomandibular fixation (MMF) to minimize mobility of flap – May divide tongue flap after 2 to 3 weeks • To help mobilize buccal or palatal flap, undermine tissue, incise periosteum on underside, remove some buccal plate or palatal bone only if necessary • Consider surgical stent as needed to protect tissue <p>Sinus precautions (4 to 6 weeks)</p> <ul style="list-style-type: none"> • No blowing nose, sneeze with mouth open, and avoid straws, smoking, or high elevation (ie, airplanes) • If repeated failed attempts to close fistula, ensure all infection is cleared to improve success <p>Preexisting sinusitis</p> <ul style="list-style-type: none"> • Prescribe postoperative antibiotic, nasal decongestant, and nasal spray for 1 to 2 weeks <ul style="list-style-type: none"> – 70% acute sinusitis bacterial species <ul style="list-style-type: none"> ◦ <i>Streptococcus pneumoniae</i> ◦ <i>Haemophilus influenzae</i> ◦ <i>Moraxella catarrhalis</i> <p>Sinus medications</p> <ul style="list-style-type: none"> • Antibiotics: Amoxicillin, amoxicillin/clauvulanic acid, ampicillin, azithromycin, clarithromycin • Antihistamines: Certirizine, diphenhydramine, fexofenadine, loratidine • Decongestants: Pseudorephedrine, triprolidine, oxymetazoline
Mandible fracture	<ul style="list-style-type: none"> • Incidence very rare after third molar extraction (0.005%) • Risk factors <ul style="list-style-type: none"> – Patient age > 40 years – Male – Atrophic jaw – Associated cyst/tumor – Osteoporosis – Inexperienced surgeon • Most occur 14 days after surgery but can occur up to 6 weeks postoperatively • Occur mostly at the angle of the mandible • Can manage with closed reduction for 4 weeks versus open reduction and internal fixation

(Iatrogenic damage to adjacent teeth/structures cont)

Nerve injury

Inferior alveolar nerve (IAN)

- Usually located buccal and slightly apical to mandibular tooth roots
- Injury incidence: 1% to 5% after third molar extraction
- 0% to 0.9% of symptoms persist after 6 months
- Radiographic signs of increased risk for IAN injury (Rood criteria)
 - Loss of IAN canal wall—cortical (white) lines
 - Third molar root darkening
 - Narrowing of IAN canal as it passes third molar root
 - Diversion of IAN canal
 - Deflection of roots
 - Bifid root apex
- Panoramic radiograph prediction of IAN exposure/injury: Fair sensitivity, but high specificity
 - High (> 90%) negative predictive value; if no high-risk findings, IAN injury unlikely
 - Poor (30% to 70%) positive predictive value; if high-risk finding, IAN injury likely
- CBCT: Very helpful in determining IAN location
- Risk factors
 - Age > 25 years
 - Female > male
 - Tooth elevation stretching nerve
 - Instrumentation near nerve
 - Socket curettage
 - Socket medications
 - Articaine blocks
- **Consider coronectomy** (Fig 3-8): Partial tooth removal, partial odontectomy, intentional partial odontectomy (IPO), or intentional root retention
 - Possible alternative to avoid IAN injury
 - Indications
 - No pathologic lesion
 - No interference with future restorations or orthodontic treatment
 - Technique
 - Do not elevate tooth; keep attached to apical blood supply
 - Carefully section crown from roots at 45-degree angle (measured buccolingually)
 - Reduce remaining tooth to 3 mm below crestal bone (allows bone formation over root fragments)
 - Thorough irrigation
 - Water-tight primary closure (decreases chance of postoperative infection)
 - No need for postoperative root canal therapy or bone graft

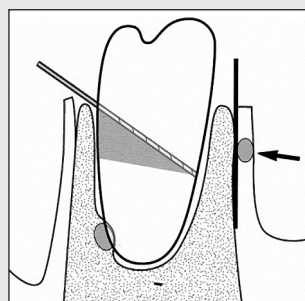


Fig 3-8 Coronectomy technique for removal of the mandibular right third molar. Note the angle of the bur set to approximately 45 degrees and the lingual retractor placed protecting the lingual nerve (arrow). The shaded area of root on the buccal side is to be removed secondarily. (Reprinted with permission from Pogrel M, Lee JS, and Muff DF. Coronectomy: A technique to protect the inferior alveolar nerve. J Oral Maxillofac Surg 2004;62:1447–1452.)

(Iatrogenic damage to adjacent teeth/structures cont)

Lingual nerve

- Sensory innervation and taste (carried from CN VII along CN V) of anterior $\frac{2}{3}$ tongue
- Anatomical variations of lingual nerve location
 - Most common: 2.5 mm medial and 2.5 mm inferior to crest of lingual plate
 - Other: 10% to 15% above crest of lingual plate
 - 25% in direct contact with lingual plate and/or attached to periosteum/third molar follicle
- Injury incidence: 0.4% to 1.5%, but 0% to 0.5% persist after 6 months
 - Range is 0% to 22% (due to anatomical variations)
 - 96% of cases have spontaneous recovery
- Highest risk: Lingually angled impactions; erosion/absence of lingual plate from infection/cyst
- Lingual plate usually thin, so do not section through crown completely during tooth removal
- If lingual plate fractures, keep periosteum attached, minimize manipulation
- Ensure flap design that avoids the nerve
- When using lingual flap, need proper flap retraction and lingual nerve protection
- Retraction may cause transient lingual nerve paresthesia

Long buccal nerve

- Branches are frequently cut (especially if aberrant path), but its effects usually are not noticed by patient

Mylohyoid nerve

- Incidence of injury up to 1.5% (likely from lingual flap retraction)

Miscellaneous

- Nerve injury from local anesthetic injection (risk 1:400 to 1:750,000)
- Cause
 - Injection into epineurium can cause hematoma and transient/permanent paresthesia
 - Reaction to local anesthetic, especially articaine

CN, cranial nerve.

Nerve terminology (Fig 3-9)

- Nerve fiber: Axon, central cell body and its extension, +/- myelination by Schwann cells
- Mechanoreceptor: Receptor excited by mechanical pressure
- Nociceptor: Receptor excited by injury/painful sensation
- Endoneurium: Surrounds group of fibers (myelinated or unmyelinated); forms fascicle
- Perineurium: Surrounds groups of fascicles
- Epineurium: Outermost layer of peripheral nerve; surrounds fascicle bundles and blood vessels and protects nerve against compressive forces
- Wallerian degeneration: Fatty degeneration of the nerve fiber

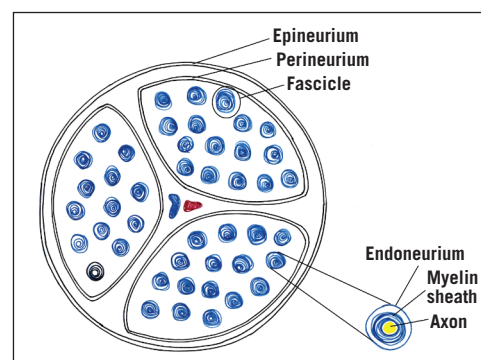


Fig 3-9 Nerve layers (illustrated by Quoc Lu.)

Classifications of nerve injury

- Seddon (3 categories) and Sunderland (5 categories, more cellular description)

	Event	Nerve sheath	Axons	Wallerian degeneration	Treatment	Possible spontaneous recovery
Seddon I: Neuropraxia (Sunderland I)	Minor compression/traction, temporary conduction blockade	Intact	Intact	No	No micro-surgery indicated	Hours to 2 months
Seddon II: Axonotmesis (Sunderland II)	Minor crush/traction	Intact	Interrupted	Partially at distal	Microsurgery only if foreign body present	2 to 4 months
Sunderland III	Moderate crush/traction	Endoneurium loss, perineurium intact	Interrupted, neuronal death	Yes	Microsurgery if no recovery in 3 to 4 months	3 to 4 months, mild/moderate permanent sensory disturbance, slow recovery
Sunderland IV	Severe crush/traction	Endoneural/perineural disruption, epineurium intact, permanent alteration in blood/nerve barrier	Neuronal loss +/- neuroma formation, possible intraneural fibrosis	Yes	Microsurgery if no recovery in 3 to 4 months	3 to 4 months, mild/moderate permanent sensory disturbance, slow recovery
Seddon III: Neurotmesis (Sunderland V)	Complete transection	Loss of endoneurium, perineurium, and epineurium	Interrupted, +/- neuroma formation	Complete transection	Need microsurgery	Unlikely; permanent sensory disturbance

Nerve repair

- Clinical testing: Map area of sensory disturbance (Fig 3-10)
- Level A (A-alpha and A-beta fibers): Mechanoreceptors
 - Brush stroke direction +/- vibration discrimination
 - Use cotton wisp
 - Normal: 1-cm area, 90%
 - 2-point discrimination
 - Use Boley gauge with 2-mm increments
 - Normal: 4 mm for IAN, 3 mm for lingual nerve
- Level B (A-beta fibers): Mechanoreceptors – Contact detection: Static light touch and pressure preception
 - Use cotton wisp or von Frey monofilaments
- Level C (A-delta [myelinated] and C fibers [unmyelinated]): Nociceptors
 - Pinprick nociception: Assess free nerve endings
 - Thermal discrimination: Hot (heated gutta percha [A-delta fibers]) versus cold (ethyl chloride [C fibers])
- If dysesthesia, use diagnostic nerve block
 - If relief, need microsurgery
 - If no relief, central process and need pharmacologic treatment
- Microsurgery repair
 - Indications (best results if < 10 weeks)
 - Complete anesthesia > 1 to 2 months
 - Profound hypoesthesia that does not improve > 3 months
 - Early dysesthesia
 - Clinical observation of transection or foreign body
 - Progressive/worsening symptoms
 - Patient not able to tolerate hypoesthesia
 - Contraindications
 - Centrally mediated pain: Treatment = medication (ie, clonazepam 0.5 mg every 8 hours, gabapentin, pregabalin)
 - Improving sensory function
 - Symptoms are acceptable to patient
 - Medically compromised patient
 - Patient age
 - Long duration since injury
- Microsurgery nerve repair techniques
 - External neurolysis: Free up nerve
 - Internal neurolysis (controversial due to possible increased scar formation): When gross change seen or evidence of fibrosis, open epineurium to decompress
 - Excision of neuroma/fibrosis and resection in 1-mm increments until healthy nerve visualized
- Options: Direct/primary neurorrhaphy; better outcomes than autogenous graft
 - Epineural repair with minimal number of 8-0 nonreactive sutures
 - Tension-free primary anastomosis (max IAN gap = 5 mm, max lingual nerve gap = 10 mm)

Examiner(s) Name: _____

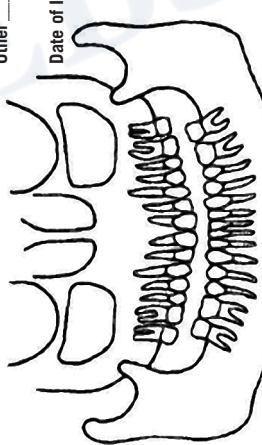
Date _____ **Patient ID#** _____ **Gender** _____

Age _____

Mechanism of Injury (check one)

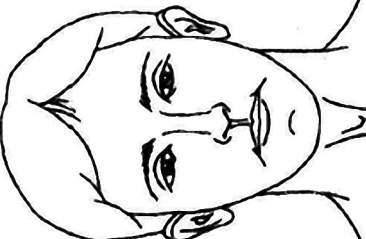
☐ Odontectomy
☐ Orthognathic
☐ Implant
☐ Needle
☐ Trauma
☐ Other _____

Right



Left

Right

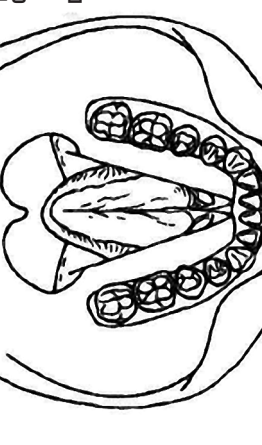


Date of Injury _____

Upper **% of**

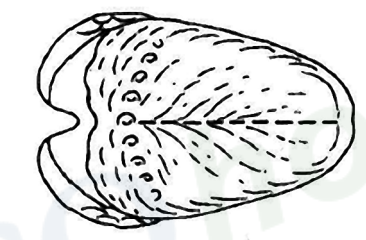
Level A	Level B	Level C
Brush stroke	ascending	Pain (+ or -)
Static 2PDT	Contact descending	Chin
	Detection threshold	Threshold (1)
		Threshold (2)
		Tolerance (1)
		Tolerance (2)

Right



Left

Right



Date of Injury _____

Upper **% of**

Level A	Level B	Level C
Brush stroke	ascending	Pain (+ or -)
Static 2PDT	Contact descending	Tongue
	Detection threshold	Threshold (1)
		Threshold (2)
		Tolerance (1)
		Tolerance (2)

Trigger

☐ painful
☐ nonpainful
☐ radiating
☐ nonradiating
☐ none

Comments: _____

Obj. rating

☐ normal
☐ mild
☐ moderate
☐ severe
☐ complete

Fig 3-10 Nerve injury mapping charts. Clinical neurosensory test forms used for reporting sensory impairment scores in patients with inferior alveolar nerve (A) and lingual nerve injuries (B). Each form is designed for recording the necessary demographic information about the patient; recording the location and type of trigger response, recording test outcomes of Levels A, B, and C; and determining the sensory impairment score based on test outcome. 2PDT, two-point discrimination test. (Reprinted with permission from Zuniga JR, Meyer RA, Gregg JM, Miloro M, Davis LF. The accuracy of clinical neurosensory testing for nerve injury diagnosis. *J Oral Maxillofac Surg* 1998;56:2-8.)

Autogenous nerve graft considerations

	Location	Diameter (mm)	Cross-section shape	Fascicular pattern	Length of harvest	Morbidity
IAN		2.4	Round	18–21		
Lingual nerve		3.2	Round	15–18		
Sural nerve (S1–S2)	Sensory to dorso-lateral foot, parallel to lesser saphenous vein	2.1	Flat	11–12	Up to 20 cm	Numb heel/lateral foot
Great auricular nerve (C2–C3)	Ascends on SCM to innervate skin behind auricle and parotid gland	1.5	Oval	8–9	1–2 cm	Numb lateral neck, posterior mandible, ear

SCM, sternocleidomastoid.

- Entubulation: Nerve grows within conduit about 1 mm/day
 - Autogenous, allogeneic, xenograft: Collagen, muscle, fascia, vein
 - Alloplastic: Polyglycolic acid, polyester, polytetrafluoroethylene
- All patients need sensory education after repair
- Prognosis: Overall success about 50%a
 - Hypoesthesia: Indicates better success than hyperesthesia
 - Painful neuroma: 70% patients improve with all surgical techniques
 - Poorer outcomes: If repair delayed > 6 months
 - Risk chronic pain syndrome: Increased if repair delayed > 1 year

Postoperative complications

Pain	Alveolar osteitis (dry socket) <ul style="list-style-type: none"> • Average incidence after mandibular third molar extraction: 3% to 5% • Possible risk factors: Age > 25 years, female gender, smoker, oral contraceptives, surgeon's experience, operation time, premature mouth rinsing, pericoronitis, bacterial contamination, poor oral hygiene • Clinical symptoms <ul style="list-style-type: none"> – Usually develops between postoperative days 2 to 5 – Throbbing, radiating pain (most common), fetid odor, bad taste • Etiology – <ul style="list-style-type: none"> Unclear – Theories <ul style="list-style-type: none"> ◦ Increased fibrinolytic activity from tissue, saliva, or bacteria and accelerated blood clot lysis before replacement by granulation tissue ◦ Anaerobic bacteria disturb wound healing; reducing bacterial load may decrease incidence of alveolar osteitis • Prevention <ul style="list-style-type: none"> – Copious intraoperative and postoperative irrigation – Preoperative chlorhexidine rinse – Antibiotic placement in extraction site (tetracycline or lincomycin)
-------------	--

(Postoperative complications cont)

	<ul style="list-style-type: none">• Treatment<ul style="list-style-type: none">– Nonsteroidal anti-inflammatory drugs (NSAIDs) as needed– Gentle irrigation with warm isotonic saline– Sedative eugenol dressing changed every 24 to 48 hours as needed (beware of reported neurotoxic effects of eugenol)– No postoperative antibiotics indicated unless overt purulence or lymphadenopathy– Do not curette/stimulate bleeding; may worsen condition and further delay healing<ul style="list-style-type: none">◦ Will destroy current healing process, and localized inflammatory process may spread to surrounding healthy bone• Prognosis<ul style="list-style-type: none">– Pain usually resolves in 3 to 5 days but can take up to 14 days <p>Temporomandibular joint (TMJ) pain</p> <ul style="list-style-type: none">• Cause: Prolonged mouth opening, jaw stretching, pressure on contralateral joint during mandibular surgery• No correlation between internal derangement and third molar removal• Prevention: Use mouth prop and provide intraoperative mandibular support• Treatment: Moist heat, soft diet, rest of joint, NSAIDs• If present > 2 weeks, consider arthrocentesis
Trismus	<p>Possible causes</p> <ul style="list-style-type: none">• Muscle spasm<ul style="list-style-type: none">– Normal occurrence peaks on day 2 and resolves by end of first week– Treatment: Similar to myofascial pain• Infection – Slow onset, swelling, fluctuance, palatal draping (pterygomandibular space involvement)<ul style="list-style-type: none">– Treatment: Antibiotics and possible incision and drainage• Local anesthetic injection – Trauma to muscles or hematoma formation in pterygomandibular space or infratemporal fossa from needle<ul style="list-style-type: none">– Increased risk with multiple injections –Treatment: NSAIDs, moist heat, physiotherapy (mouth opening and lateroexcursive exercises)• Swelling/edema – Expected after surgery; usually peaks in 48 to 72 hours; resolves in 5 to 7 days – Preoperative intravenous corticosteroids may minimize swelling; may also use oral steroids for 1 to 2 days postoperatively<ul style="list-style-type: none">– Most common steroids used<ul style="list-style-type: none">◦ Dexamethasone 4 to 12 mg intravenous preoperatively◦ Methylprednisolone 125 mg intravenous preoperatively◦ Both steroids are almost pure glucocorticoids (versus mineralocorticoids)◦ They are least depressing on leukocyte chemotaxis◦ High-dose use for short term has minimal side effects◦ Contraindications: Peptic ulcer, active infection, psychosis– Other treatments<ul style="list-style-type: none">◦ Ice packs to face intermittently for first 24 hours helps with comfort but does not significantly diminish edema◦ Heat to face intermittently after 24 hours to help eliminate swelling

(Postoperative complications cont)

Periodontal defect	Risk factors <ul style="list-style-type: none"> • Age > 25 years (30% risk of increased pocket depth) • After extraction, new bone formation is not radiographically evident until 6 to 8 weeks later • Site appears different from adjacent alveolar bone for about 4 to 6 months
Ecchymosis/hematoma	<ul style="list-style-type: none"> • Occurs frequently, especially dramatic in elderly patients (increased capillary fragility, decreased tissue elasticity) • Extensive if poor hemostasis intraoperatively and/or immediately postoperatively • Treatment: Intermittent ice packs for 24 hours, then intermittent moist heat • If large hematoma: Evacuate with large bore needle, postoperative antibiotics
Wound dehiscence	<ul style="list-style-type: none"> • Possible causes: Compromised blood supply to site (tissue tearing, over-retraction, sutures too tight) • Treatment: <ul style="list-style-type: none"> – Let heal by secondary intention (do not resuture wound) – Reduce any sharp/rough edges of exposed bone with rongeur/bone file – If large exposed area of nonvital bone, allow separation from base on its own; remove when loose
Postoperative wound infection	<ul style="list-style-type: none"> • Extraction: Clean-contaminated procedure <ul style="list-style-type: none"> – Prophylactic antibiotics not indicated – Postoperative infection more dependent on operator technique (versus nonantibiotic use) • Average postoperative infection rate for experienced surgeon in normal healthy patients = 1% to 5% <ul style="list-style-type: none"> – Treat with debridement, irrigation, antibiotics • Pre- and postoperative chlorhexidine rinses (Peridex, 3M) may help decrease infection and alveolar osteitis
Subperiosteal abscess	<ul style="list-style-type: none"> • Caused by debris under mucoperiosteal flap • Occurs 2 to 6 weeks postoperatively • Treatment <ul style="list-style-type: none"> – Debridement, irrigation, antibiotics – Consider radiograph if does not resolve
Interstitial emphysema	<ul style="list-style-type: none"> • Air forced into connective tissue or fascial planes under pressure • Conventional dental handpiece contraindicated when cutting in proximity to an open socket or a flap due to expelled forced air • Diagnosis: Sudden facial swelling, palpable crepitus, +/- pain – May dissect through fascial planes into mediastinum – If air in venous system (ie, pterygoid or intraosseous venous plexus), air embolus to heart can lead to cardiac arrest • Treatment <ul style="list-style-type: none"> – Antibiotics (ie, cephalosporin); prevents secondary infection from possible oral bacterial nidus – Systemic corticosteroids may help faster resolution – Resolves in 7 to 10 days

Soft Tissue and Preprosthetic Procedures

Intraoral Flap Design

Envelope flap +/- releasing incision	<ul style="list-style-type: none"> • Indication: Workhorse flap design for majority of procedures • Vertical releasing incisions – Releases tension on sutures; avoids pull through tissue, wound dehiscence/periodontal defects <ul style="list-style-type: none"> – Flap base should be broader than flap apex (for adequate blood supply) – Should run obliquely (versus straight) – Should cross free gingival margin at tooth line angles • Contraindicated areas for releasing incisions <ul style="list-style-type: none"> – Canine eminence: Risk of wound dehiscence – Mental foramen: Risk of injuring mental nerve – Mandibular lingual surface: Lingual nerve injury – Through muscle attachments: Wound dehiscence – Directly through dental papilla: Gingival recession • Reposition flap margins over sound bone
Semilunar flap	<ul style="list-style-type: none"> • Slight half-moon horizontal incision in attached gingiva (Fig 3-11) • Indications: Root tip removal access, apicoectomy, access to apical area of implant without disruption of attached gingiva • Avoids interdental papillae when only small area of access needed; easy reflection but restricted access
Submarginal flap	<ul style="list-style-type: none"> • Horizontal incision in attached gingiva with scalloped margins; 1 to 2 releasing incisions at obtuse angles (Fig 3-12) • Indications: Access to more than one tooth area without disruption of attached gingiva • Requires minimum of 4 mm attached gingiva and good periodontal health • Avoids interdental papillae to maintain esthetics; mostly used in anterior maxilla but sometimes produces scars

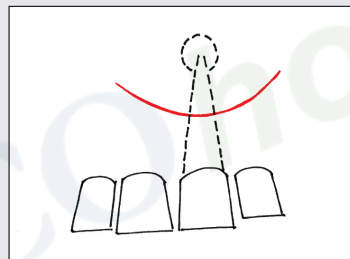


Fig 3-11 Semilunar flap (illustrated by Quoc Lu).

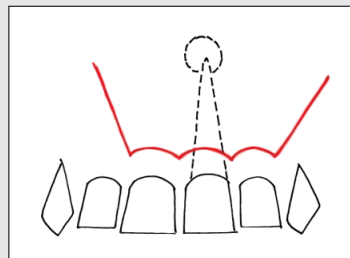


Fig 3-12 Submarginal flap (illustrated by Quoc Lu).

Preprosthetic Surgery

Blood supply of edentulous mandible

- Inferior alveolar vessels shrink with age: Primary blood supply moves centripetally (center seeking) from periosteum
 - In dentate mandible blood supply moves centrifugally (center fleeing) from inferior alveolar artery
- Tip: Avoid excessive periosteal elevation, which compromises blood supply

Bone resorption patterns

- Edentulous maxilla: Bone resorbs more quickly and dramatically than in mandible
- Maxillary ridge resorbs on lateral and inferior sides—crest moves posteriorly and superiorly; possibly from lack of muscle attachments (less functional stimulus)
- Edentulous mandible: Crest moves anteriorly with decreasing height and width
- Collapsing vertical dimension; mandible autorotates forward
- Creates skeletal pseudo-Class III relationship

Combination syndrome

- Edentulous anterior maxilla undergoes excessive resorption from opposing forces of anterior mandibular teeth (Fig 3-13)



Fig 3-13 Combination syndrome.

Cawood and Howell classification of edentulous ridge

I	Dentate ridge
II	Ridge immediately postextraction
Modification	No defect, buccal wall, or multiwall defect
III	Well-rounded ridge with adequate height and width
IV	Knife-edge ridge with adequate height but inadequate width
V	Flat ridge with inadequate height and width
VI	Depressed ridge with basilar bone loss

Tuberosity reduction

- Minimal distance required between maxillary tuberosity crest and mandibular retromolar pad: 10 mm
- If inadequate clearance, vertical dimension is decreased
- Also check for horizontal prominence and undercuts
- **Technique**
 - Elliptical subperiosteal wedge-shaped incision
 - Undermine buccal and palatal tissues subperiosteally
 - Thin flaps by removing excess submucosal tissue
 - Remove any excess mucosa and suture wound

Frenectomy

- Frenum: Thin band of fibrous tissue covered with mucosa that extends from lip/cheek/tongue to alveolar ridge
- Tips
 - Must excise fibrous band and mucosal attachment; otherwise, high recurrence rate
 - If narrow attachment, simple excision or Z-plasty
 - If wide attachment, vestibuloplasty

Simple excision: Labial frenectomy

- Infiltrate local anesthetic into surrounding area (not directly into frenum, which distorts anatomy)
- Evert lip to clarify anatomy
- Make elliptical supraperiosteal incision around frenum
- Use scissors to sharply excise mucosa and underlying connective tissue band
- Leave periosteum on alveolar ridge
- Undermine labial tissue margins and reapproximate with interrupted sutures passed through periosteum (preserves length and prevents hematoma formation)
- Labial attached gingiva defect heals via secondary intention

Z-plasty: Labial frenectomy

- Use if alveolar height is questionable and need to maintain vestibular depth
- Make elliptical supraperiosteal incision around frenum
- Undermine flaps; make two releasing incisions at a 45-degree angle in relation to the initial vertical incision
- Rotate flaps to make horizontal closure of original vertical incision

Lingual frenectomy

- Treats ankyloglossia or tongue-tie
- Tissues involved: Mucosa, connective tissue, and superficial genioglossus muscle
- Administer bilateral lingual nerve blocks and local infiltration in anterior floor of mouth
- Retract tongue with midline suture at tip
- Excise mucosa and fibroelastic band from ventral tongue attachment to insertion on attached lingual alveolar gingiva to release ventral tongue
- Avoid Wharton ducts and superficial blood vessels
- Have patient protrude the tongue
- Transversely transect a portion of the genioglossus muscle if tongue length inadequate
- Undermine all incision edges
- Prevent floor-of-mouth hematoma with good hemostasis
- Close incision in linear fashion with interrupted or running suture and buried knots
- May also use V-Y closure, Z-plasty, or back-to-back Z-plasty to increase tongue length and mobility (Fig 3-14)

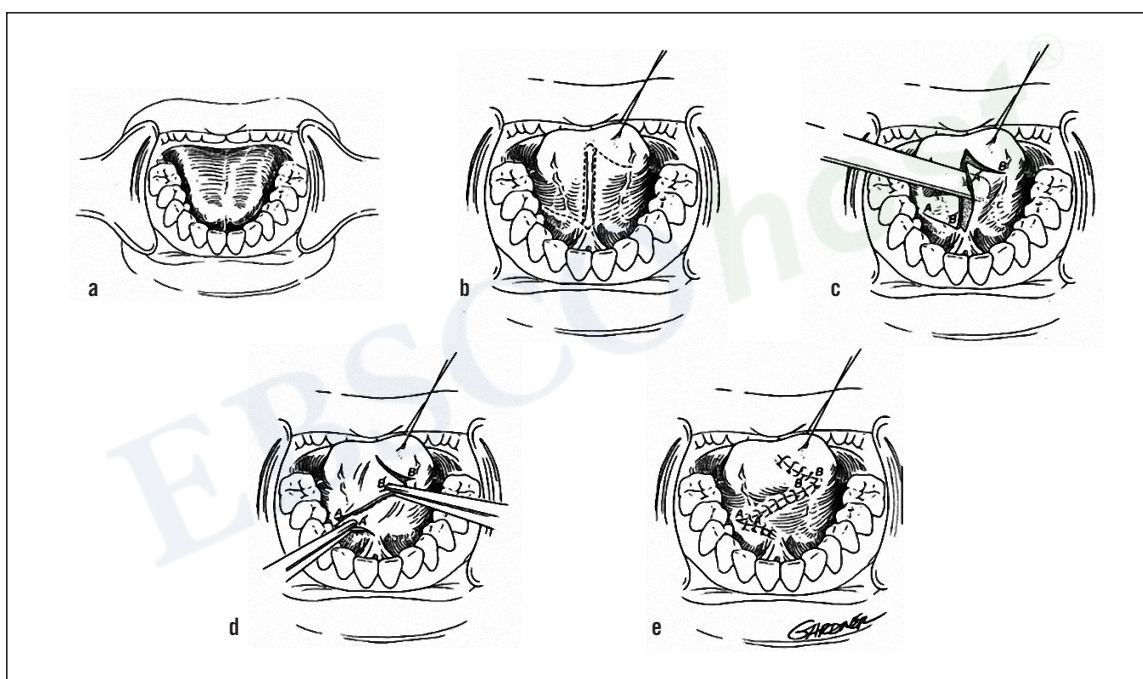


Fig 3-14 Z-plasty for lingual frenectomy. (a) Preoperative lingual frenum. (b) Proposed incisions for a lingual frenectomy with Z-plasty closure. (c) Undermining wound margins. (d) Transposition of Z-plasty flaps. (e) Final closure. (Reprinted with permission from Digman SW. Pediatric dentoalveolar surgery. In: Fonseca RJ, Marciani RD, Turvey TA [eds]. Oral and Maxillofacial Surgery, ed 2. Vol 1: Anesthesia and Pain Control, Dentoalveolar Surgery, Practice Management, Implant Surgery. Philadelphia: Saunders, 2009:176.)

Vestibuloplasty

- Goals
 - Increase sulcus depth to extend denture-bearing area and improve retention
 - Increase attached alveolar tissue for stable denture base
- To avoid chin sag: Maintain minimum of 10 mm of mentalis muscle attachment

Maxillary submucosal vestibuloplasty

- **Indications** – Good alveolar bone height and contour
– Shallow vestibule or high muscle attachments make denture unstable
- **Technique** – Check for adequate labiovestibular depth
– Place tongue blade/mouth mirror into depth of maxillary vestibule; ensure no upper lip distortion/inversion – If depth inadequate or if lip distortion, perform Kazanjian or transpositional vestibuloplasty instead – If adequate depth and no distortion, make vertical midline incision – Use scissors to bluntly undermine mucosal layer supraperiosteally on anterior maxilla – Bluntly dissect supraperiosteal muscle fibers and soft tissue attachments and retract or remove to increase direct contact between mucosa and periosteum
– Close incision and insert denture with extended peripheral margins or surgical stent to maintain maximum vestibule depth
- Maintain denture or stent for 2 weeks; allows mucosal adherence to underlying periosteum
- After 1 month do definitive denture relines

Kazanjian flap vestibuloplasty

- **Indication:** In maxilla or mandible when submucosal vestibuloplasty contraindicated (ie, inadequate labial vestibular depth)
- **Technique** (Fig 3-15)
– Horizontal incision in labial mucosa at junction of attached and unattached gingiva; create supraperiosteal flap in unattached mucosa
– Reposition flap apically by suturing mucosal edge to periosteum at point of greatest vestibular depth
– Allow healing by secondary epithelialization or place split-thickness skin graft or AlloDerm (LifeCell) to shorten healing time
– Place relined denture for 2 to 3 weeks to increase patient comfort and mold/adapt underlying soft tissues/skin graft

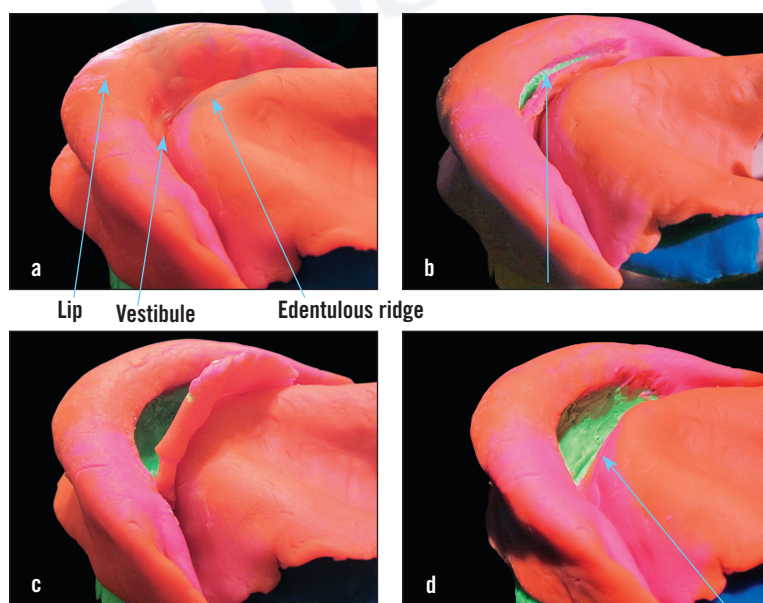


Fig 3-15 Mandibular Kazanjian flap vestibuloplasty. (a) Preoperative situation. (b) Inner lip incision on mucosa (arrow). (c) Lip mucosa is reflected to reveal submucosa (green). (d) Mucosal flap edge sutured down to create new vestibule depth. Lip submucosa heals secondarily.

Mandibular transpositional flap (“lip-switch”) vestibuloplasty

- **Technique** (Fig 3-16)
 - Make a horizontal incision on inner surface of lower lip
 - Continue suprapariosteal mucosal dissection superiorly up to alveolar ridge crest
 - Incise periosteum on alveolar crest and create an inferiorly based periosteal flap
 - Transpose and suture periosteal flap to denuded labial submucosa
 - Suture mucosal flap to periosteum at depth of vestibule to cover anterior mandible
 - Place relined denture for 2 to 3 weeks to improve patient comfort and mold/adapt underlying soft tissues

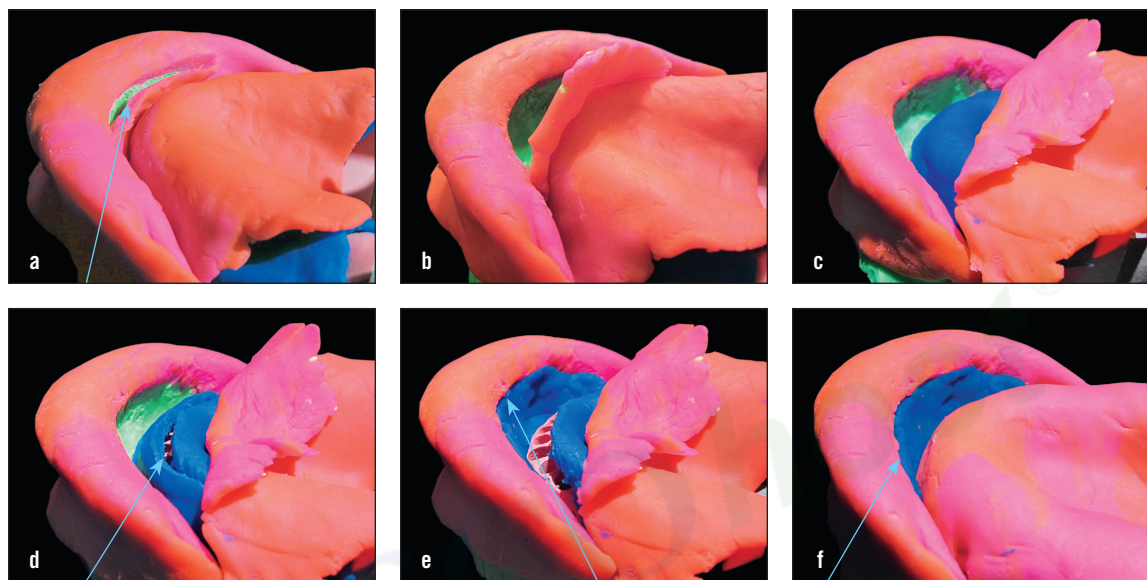


Fig 3-16 Transpositional flap “lip-switch” vestibuloplasty. (a) Incision on inner lip (arrow). (b) Mucosal reflection to reveal submucosa (green). (c) Further undermining to reveal alveolar ridge periosteum (blue). (d) Incision on alveolar ridge crest (arrow). (e) Undermine and suture cut edge of periosteum to raw mucosal edge of inner lip (arrow). (f) Suture edge of cut mucosa down to new vestibule depth (arrow).

Mandibular vestibuloplasty and floor-of-mouth lowering

- **Indication:** Extensive lack of vestibular depth (need at least 15-mm mandibular height)
- **Technique**
 - Make a crestal incision and create suprapariosteal labiobuccal and lingual mucosal flaps
 - Sharply dissect mylohyoid and genioglossus muscles from insertions (release no more than half of superior part)
 - Place submandibular sutures to hold mucosal flaps in an inferior position
 - Insert split-thickness skin graft in denture or surgical stent and place on denuded periosteum
 - Use circummandibular sutures or wires to hold denture or stent in place

Tuberoplasty

- **Indication:** Need to deepen hamular notch to increase palatal seal
- **Disadvantage:** Limited predictability/success
- **Technique**
 - Make a vertical incision behind the tuberosity to expose the inferior aspect of the pterygoid plate
 - Fracture pterygoid plate from maxilla with curved osteotome; separate from tuberosity and displace posteriorly
 - Suture tissue to desired depth to create new hamular notch area

Alternative preprosthetic considerations

- If healthy but supraerupted maxillary posterior teeth impinging on mandibular teeth or alveolar ridge
 - Reposition superiorly using maxillary posterior segmental osteotomy
- If severely atrophic maxilla but high palatal vault
 - Le Fort I osteotomy; reposition maxilla anteriorly and inferiorly; place interpositional bone graft
- If atrophic maxilla but low palatal vault
 - Onlay graft of rib or horseshoe-shaped iliac bone (corticocancellous bone)
 - Usually need secondary soft tissue procedure

Treatment of Denture-Associated Pathology

Maxillary torus palatinus	<ul style="list-style-type: none">• Indications for removal<ul style="list-style-type: none">– Very large and fills palatal vault– Interferes with denture construction– Traumatized mucosa with exposed bone– Speech interference– Bothers patient psychologically• Technique – Fabricate stent ahead of time on study cast – Inject local anesthetic under thin palatal tissue over torus for hydrodissection – Double-Y or I incision over torus midline; subperiosteal flap elevation – Score torus multiple times with bur; cleave small portions with osteotome and mallet (do not fracture into nose)<ul style="list-style-type: none">– Use large bur/bone file to smooth surface; irrigate; close wound with horizontal mattress sutures– Place stent; secure with undercuts, circumdental wires, or screws
Torus mandibularis	<ul style="list-style-type: none">• Indications for removal: When would interfere with full or partial denture construction• Technique<ul style="list-style-type: none">– Inject local anesthetic under thin lingual tissue over torus for hydrodissection – Crestal (edentulous area) or lingual gingival sulcular incision (no releasing incisions due to presence of lingual nerve); subperiosteal flap elevation– Use fissure bur to score top of torus; place retractor beneath torus to prevent displacement into floor of mouth; cleave with mallet and osteotome– Use large bur/bone file to smooth bone; close wound– Place gauze pack under tongue as pressure dressing for at least 30 minutes to prevent hematoma
Epulis fissuratum (fibrous inflammatory hyperplasia)	<ul style="list-style-type: none">• Etiology: Chronic irritation from poor-fitting denture• Treatment – Surgical excision or cryosurgery (preferably with CO₂ laser)<ul style="list-style-type: none">– If large lesion, to avoid vestibule obliteration, undermine adjacent mucosa and reposition mucosal flap as in a Kazanjian vestibuloplasty (see page 104)– Temporarily reline denture with tissue conditioner and use as stent– Better fitting/new denture

(Treatment of Denture-Associated Pathology cont)

Inflammatory papillary hyperplasia	<ul style="list-style-type: none">• Etiology: Possible fungal infection (<i>Candida</i>), mechanical irritation, poor oral hygiene, smoking• Treatment<ul style="list-style-type: none">– Antifungicides (nystatin, clotrimazole troche) and soak dentures in very dilute bleach– If extensive, suprapariosteal excision: Large curette, electrocautery, mucoabrasion, acrylic bur, cryosurgery, laser– Avoid palatal bone exposure– Reline denture with tissue conditioner– Possibly need better fitting/new denture
---	--

Odontogenic Infections

Natural Course

Stage	Duration (days)	Pain/swelling	Skin	Fluid	Bacteria
Early: Induration	0–3	Mild pain, diffuse swelling	Normal to slightly red	None	Aerobic
Second: Cellulitis	3–5	Severe pain, large diffuse swelling	Firm, erythematous	Minimal to moderate serosanguinous fluid	Mixed aerobic/ anaerobic
Last: Abscess	> 5	Severe, localized pain/ swelling	Erythematous, tender to palpation	Fluctuance	Anaerobic

Bacteriology

- Symbiosis between aerobic and anaerobic pathogens is the major cause of serious infection
 - Aerobes start early and cause tissue hypoxia and acidosis; creates environment for anaerobes
 - Anaerobes destroy tissue and cause pus formation, usually takes > 48 hours to culture
- Most common pathogens: Aerobic only (7%), mixed (60%), anaerobic only (33%)
 - Gram-positive cocci: *Streptococcus viridans*
 - Gram-negative rod/bacilli (takes > 48 hours to culture): *Bacteroides* (causes foul smell), *Fusobacterium*

Fascial Spaces

- Primary spaces
 - Submandibular space (below mylohyoid)
 - Sublingual space (above mylohyoid)
 - Vestibular space
 - Buccal space
 - Canine space (maxilla only)
 - Submental space
- Secondary spaces
 - Masticator space (includes pterygomandibular, masseteric, and temporal spaces)
 - Infratemporal space
 - Lateral pharyngeal space
 - Retropharyngeal space
 - Prevertebral space
 - Danger space 4

Radiography

- Clinical examination for pus has 45% false negative rate
- Lateral neck film
 - At the level of C2, the distance from the anterior surface of the vertebrae to the posterior border of the airway should be 7 mm or less (regardless of age)
 - At C6, the distance should be 14 mm or less (< 15 years) or 22 mm (> 15 years)
 - False negative rate is as high as 30%
- CT scan with intravenous contrast has 5% false negative rate
 - Shows “ring” enhancing lesion and abscess collection

Indications for Inpatient Treatment

- Compromised host defenses
- Rapid, progressive infection
- Secondary fascial space involvement
- Temperature over 101°F
- Severe trismus (< 10 mm)
- Toxic appearance
- Difficulty swallowing or breathing

Surgical Incisions for Drainage

Severe infections may require multiple incisions and drainage sites (both intraoral and extraoral).

Infection location	Incision and drainage locations*
Vestibular spaces	Intraoral (vestibular) incision
Buccal space	Mandibular vestibule or most dependent part of cheek swelling
Temporal space	1 cm posterior to temporal hairline and cephalic to the helix
Submandibular space	Submandibular incision 2 cm below the lower border of the mandible
Sublingual space	Anterior floor of mouth
Submental space	Below chin proximal to the lingual surface of the anterior mandible
Pterygomandibular space	Vertical incision between anterior border of mandible and medial pterygoid muscle or extraoral incision 2 cm below lower border of mandible
Lateral pharyngeal space	Extraoral submandibular incision 2 cm below lower border of mandible; introral incision subperiosteal sulcular incision lingual to posterior molars and slightly medial
Retropharyngeal spaces	2-cm vertical incision at the anterior border of sternocleidomastoid muscle (begin at the level of hyoid bone) and use a finger dissection
*Dependent location for drainage, if possible.	

Antibiotic

Commonly used antibiotics for oral and maxillofacial infections

	Mechanism of action	Remarks
Penicillin,*† amoxicillin,* and ampicillin†	Inhibits bacterial cell-wall formation	Good first line choice, but many pathogens are resistant to penicillin (beta-lactamase)
Augmentin (amoxicillin and clavulanic acid),* zosyn (piperacillin and tazobactam),† and unasyn (ampicillin and sulbactam)†	<ul style="list-style-type: none">• Inhibits bacterial cell-wall formation• Clavulanic acid, sulbactam, and tazobactam serve as beta-lactamase inhibitors	Indicated when penicillin fails to provide clinical improvement or beta-lactamase-producing organisms are suspected
Cephalexin* and cefazolin†	<ul style="list-style-type: none">• First-generation cephalosporin• Inhibits bacterial cell-wall formation	<ul style="list-style-type: none">• 10% cross-reactivity with penicillin• Contraindicated for penicillin-allergic patients

(Commonly used antibiotics for oral and maxillofacial infections cont)

	Mechanism of action	Remarks
Clindamycin ^{*†}	<ul style="list-style-type: none">• Inhibits protein synthesis by binding to 50S ribosomal subunit	<ul style="list-style-type: none">• No adjustment needed for both hepatic- or renal-compromised patients• Alternative for patients with penicillin allergy• Excellent abscess penetration
Azithromycin ^{*†} and clarithromycin	<ul style="list-style-type: none">• Inhibits protein synthesis by binding to 50S ribosomal subunit• Macrolide antibiotic family	<ul style="list-style-type: none">• Less gastrointestinal upset compared to erythromycin• Good coverage against <i>Haemophilus influenza</i> (sinus infection)
Ciprofloxacin, levofloxacin, and moxifloxacin	<ul style="list-style-type: none">• DNA gyrase inhibitor• Fluoroquinolone antibiotic family	<ul style="list-style-type: none">• Ciprofloxacin has limited oral flora coverage (anaerobic organisms and <i>Streptococcus</i>)• Good coverage for <i>Staphylococcus</i> (skin infection)• Contraindicated for pediatric population (spontaneous tendon rupture)
Metronidazole ^{*†}	<ul style="list-style-type: none">• Not well understood• Forms cytotoxic particles that interact with bacterial DNA and eventually lead to DNA strand breakage	<ul style="list-style-type: none">• Anaerobic coverage only• Excellent cerebrospinal fluid penetration• First line therapy against pseudomembranous colitis
Vancomycin [†]	Inhibits cell wall formation	<ul style="list-style-type: none">• Narrow therapeutic window (requires blood level measurement)• Should reserve for methicillin-resistant <i>Staphylococcus aureus</i> (MRSA)• Oral form only is used for pseudomembranous colitis
[*] Oral. [†] Intravenous.		

Associated Major Infections

Pseudomembranous Colitis

- Superinfection of *Clostridium difficile* due to recent antibiotic usage
 - Can be any antibiotic but most commonly associated with clindamycin, ampicillin, and cephalosporins
- **Risk factors**
 - Advanced age
 - Female gender
 - History of inflammatory bowel disease
 - History of renal disease
- **Clinical manifestations**
 - Profuse watery diarrhea
 - Abdominal cramping
 - Fever
 - Elevated white blood cell count

- **Diagnosis**
 - *C. difficile* culture from stool sample
 - Stool testing: *C. difficile* toxin assay
 - Sigmoidoscopy
- **Treatment**
 - Hydration
 - Discontinue offending antibiotic
 - Metronidazole (oral or intravenous) or vancomycin (oral form only)

Cavernous Sinus Thrombosis

- Vascular thrombosis in cavernous sinus secondary to contiguous spread of orofacial infections in retrograde fashion (dental abscess, sinusitis, nasal furuncle)
- Cavernous sinus contents
 - Cranial nerve (CN) III, IV, V1, V2, VI
 - Internal carotid artery
- Routes to cavernous sinus
 - Anterior facial vein to superior ophthalmic vein: Dangerous zones—upper lip, tip of nose, and medial cheeks
 - Deep facial vein through pterygoid plexus
- **Clinical manifestations**
 - Unilateral periorbital edema
 - Headache
 - Photophobia
 - Proptosis
 - CN palsies (III, IV, V, and VI)
 - First sign of thrombosis: Abducens paresis (loss of CN VI—lateral gaze)
- **Diagnosis**
 - Clinical examination and imaging
 - Magnetic resonance imaging using flow parameters is more sensitive than a CT scan
 - Venous wall thickening
 - Deformity of the internal carotid artery within the cavernous sinus
- **Treatment**
 - Antibiotic intravenously 6 to 8 weeks: Broad-spectrum intravenous antibiotic, initially
 - Surgical debridement or incision and drainage of source
 - Anticoagulation and steroid use are controversial

Cervicofacial Necrotizing Fasciitis

- Fulminant subcutaneous infection with spread and necrosis along fascial planes
- Death can occur within 48 hours
- Two types
 - Type 1: Mixed aerobic and anaerobic (most common)
 - Type 2: *Streptococcus pyogenes* and *S aureus*

- **Risk factors**
 - Diabetes
 - Hypertension
 - Obesity
 - Alcohol/drug use
- **Clinical presentation**
 - Acutely and severely ill patient with a high fever
 - Overlying skin may be exquisitely tender, edematous, and erythematous
 - Soft tissue crepitation from gas-producing bacteria (type II only)
 - As necrosis progresses, skin becomes pale and dusky
 - Subcutaneous tissues are pale and edematous, with fat liquefaction and “dishwater” drainage
- **Treatment**
 - Medical: Broad-spectrum antibiotic, fluid replacement
 - Airway: Intubation or tracheostomy
 - Surgery: Multiple aggressive necrotic tissue debridements (the surgical wound should be left open for continued wound care and packed with antimicrobial-soaked gauze)

Actinomycosis

- Infectious disease caused by anaerobic *Actinomyces* species (*A israelii*)
- Disease is characterized by the formation of multiple painful abscesses, which often contain sulfur-like granules
- Chronic disease can lead to multiple facial fistulae
- **Treatment**
 - Long-term high-dose antibiotic (oral penicillin for 6 weeks)
 - Surgery for debridement and removal of fistulous tracts

Oral Candidiasis

- Most common fungal infection in the mouth
- Pathogen: *Candida albicans*
- Three clinical subtypes
 - Pseudomembranous
 - Most common type
 - White coating that can easily be wiped off, leaving raw surface
 - Asymptomatic
 - Erythematous (atrophic)
 - Red and painful (burning mouth)
 - Clinical presentation
 - Angular cheilitis
 - Denture-related stomatitis
 - Median rhomboid glossitis
 - Hyperplastic
 - White coating that cannot be removed
 - Asymptomatic
 - Diagnosis by biopsy

- **Risk factors**
 - Immunocompromise
 - Recent antibiotic use
 - Poor denture hygiene
 - Xerostomia
 - Smoking
 - Malnutrition
- **Treatment**
 - Nystatin oral suspension 100,000 U/mL: 5 mL four times a day for 14 days
 - Clotrimazole troches: 10 mg four times a day for 14 days

Zycomycosis (Mucormycosis)

- Fungal infection that has tendency to invade into the vascular network, which results in thrombosis and necrosis of the surrounding tissues
- Seen mostly in immunocompromised patients (ie, diabetes, HIV)
- Two types
 - Rhino-orbital-cerebral form—aggressive
 - Rhinomaxillary form—less aggressive
- **Diagnosis** by tissue biopsy: Large, broad nonseptate hyphae with right-angle branching
- **Treatment**
 - Multiple surgical debridements
 - Intravenous amphotericin B

Noma (Orofacial Gangrene)

- A rapidly progressive, polymicrobial, opportunistic infection that can lead to painless tissue degradation and permanent disfigurement
- Mostly seen in Africa and Asia
- Affects children < 12 years old
- 80% mortality rate
- Most commonly associated pathogens
 - *Fusobacterium necrophorum*
 - *Prevotella intermedia*
- **Risk factors**
 - Malnutrition
 - Poor oral hygiene
 - Immunodeficiency disease
- **Treatment**
 - Antibiotic (intravenous ampicillin)
 - Surgical debridement and incision and drainage
 - Nutritional improvement
 - Reconstructive surgery

Temporary Anchorage Devices (TADs)

Indications

- Alternative to undesired tooth mobility/movement when teeth used for orthodontic anchorage
- Retraction, protraction, uprighting, intrusion
- Also used for mild to moderate skeletal discrepancies
- Since 2006, US Food and Drug Administration (FDA) cleared for patients > 12 years

Contraindications

- Absolute contraindication: Allergy to titanium alloys
- Relative contraindications
 - Heavy tobacco use
 - Severe osteoporosis
 - Uncontrolled immune/metabolic disorders (ie, uncontrolled diabetes)
 - Bisphosphonate use

Advantages

- Less reliant on patient compliance or existing dentition
- Can be used to deliver constant or intermittent force
- Simple placement and removal
- High patient acceptance (minimizes use of interarch elastics and bulky orthodontic gear)
- Excellent success rate

Types of TADs

There are two types of TAD: Miniscrews and miniplates (Fig 3-17).

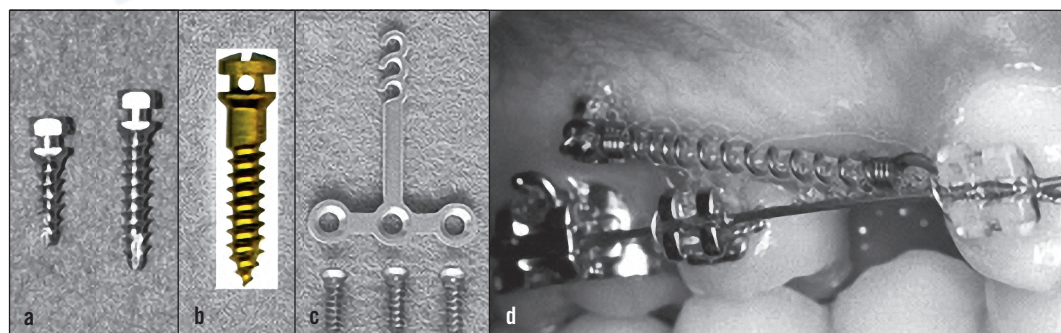


Fig 3-17 Types of TAD. (a and b) Type A titanium screws, 2.0 mm in diameter with variable lengths. (c) Miniplate with screws, 2 mm in diameter and 5 mm in length. (d) Clinical use of a type A screw. (Parts a, c, and d modified with permission from Kuroda S, Sugawara Y, Deguchi T, et al. Clinical use of miniscrew implants as orthodontic anchorage: Success rates and postoperative discomfort. Am J Orthod Dentofacial Orthop 2007;131:9–15. Part b courtesy of KLS Martin LP.)

Miniscrews

- Placed in dense cortical bone; no osseointegration
- **Characteristics**
 - Screw diameter: At least 1.5 mm
 - Length: 5 to 12 mm
 - Thread pitch (usually 0.6 mm); helps mechanical engagement of threads to cortical bone
 - Screw body: Engages thick alveolar bone apically
 - Screw head: Coronal emergence through attached gingiva (minimizes irritation, infection, gingival overgrowth)
 - Place at 30- to 45-degree angle to occlusal plane; engages maximum bone and provides ready position for orthodontic use
- **Advantages**
 - May place via flapless approach +/- soft tissue punch with either drill or by hand
 - May load immediately after placement but preferably should wait 2 to 3 weeks
- **Disadvantage**
 - Reports of 15% to 30% failure (versus miniplate)

Miniplates

- **Characteristics**
 - Low profile, similar to titanium fracture plates
 - At one end, 2 to 5 holes to fix to bone with monocortical screws (avoids tooth roots)
 - At other end, transmucosal emergence through attached gingiva with orthodontic tube, button, notch, and/or groove
 - Requires L-shaped full-thickness mucoperiosteal flap for placement and removal (Fig 3-18)
 - The horizontal incision is placed about 1 mm away from mucogingival junction within attached gingiva
 - Allows transmucosal emergence within attached gingiva (minimizes irritation, infection, gingival overgrowth)
- **Advantages**
 - More three-dimensional stability than with miniscrew, which allows more orthodontic force
 - Reports of < 5% failure (versus 15% to 30% with miniscrews)
- **Disadvantages**
 - Usually need to wait 10 to 14 days after placement to load; start with light forces and gradually increase
 - If start to load too late (> 3 weeks), may become mobile from lack of constant loading forces

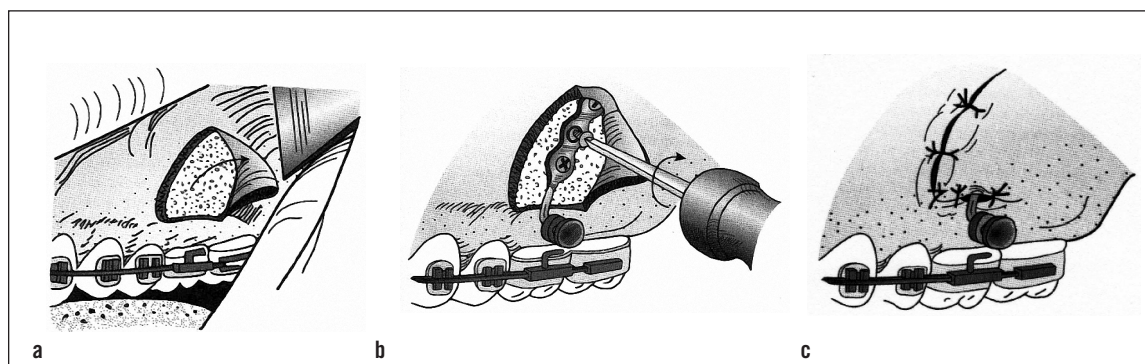


Fig 3-18 Placement of TAD (bone plate) 1 mm from transition line—transmucosal emergence through fixed gingiva. (a) Maxillary anchor plate procedure. A small L-shaped incision is used to allow for placement of an anchor plate. (b) Three screws or more are placed with appropriate positioning for the indicated orthodontic mechanics. (c) Closure is achieved with resorbable suture. (Reprinted with permission from Costello BJ, Ruiz RL, Petrone, Sohn J. Skeletal anchorage for orthodontics. In: Fonseca RJ, Marciani RD, Turvey TA [eds]. Oral and Maxillofacial Surgery, ed 2. Vol 1: Anesthesia and Pain Control, Dentoalveolar Surgery, Practice Management, Implant Surgery. Philadelphia: Saunders, 2009:233.)

Ideal Location of Placement

- Thick cortical bone: Ensure enough bone volume close to area of needed anchorage
 - Maxilla: Zygomaticomaxillary or pyriform buttress (between lateral incisor and canine), hard palate, or alveolar bone between tooth roots
 - Mandible: Symphysis, posterior body at retromolar pad, ramus, or alveolar bone between roots—especially between canine and lateral incisor
- Stay clear of adjacent vital structures
 - Minimum 2 mm away from tooth roots and vital structures
 - Minimum 1 cm away from incisive canal
 - Avoid midpalatal suture: May affect transverse maxillary growth
 - Use paramedian (parasutural) placement; avoid perforation through thin bone into maxillary sinus

Perioperative Considerations

- Insert TAD carefully to avoid shear or fracture
- Postoperative radiograph to confirm placement
- Use preoperative and postoperative antibiotics and chlorhexidine rinses
- Maintain good oral hygiene
- Labial mucosal irritation peaks around 10 days postoperatively; recommend use of wax coverage as needed

Orthodontic Techniques

- Direct: Anchor for orthodontic forces
- Indirect: Stabilize whole unit of teeth used for anchorage
- May attempt immediate load of 50- to 200-g orthodontic force allowed but preferably wait 2 to 3 weeks for stability prior to loading
- Excess torque > 250 g decreases stability; higher risk of failure
- Up to 30% relapse for intrusion of molars (depends on force amount, treatment period, degree of intrusion)

Possible Complications

- Infection, damage to adjacent structures (teeth/nerve)
- Loosening/migration of TAD
- Maxillary sinus perforation
- Hardware fracture
- Peri-implant mucositis (major cause of TAD failure)

Apicoectomy

Indications

- Resection of undebrided/unobtured portion of root and apical curettage
- When conventional root canal retreatment unfeasible via coronal approach (calcification, severe root curvatures)
- Restorative concerns (crown, post and core); removal may be difficult and/or cause root fracture

- Horizontal root fracture, especially from trauma, leading to apical pulp necrosis
- Irretrievable material in canal (broken file, restorative material); need to remove with root apex
- Iatrogenic failure (ledge, overfill, perforation)
- Large lesions that do not resolve after root canal therapy

Contraindications

- When able to perform root canal therapy or retreatment via conventional approach
- Anatomical barriers to apex (ie, maxillary sinus, zygomatic buttress, prominent chin, mental foramen)
- Short roots; may decrease tooth stability
- Systemic health issues

Procedure

1. Incision: Sulcular (best access), semilunar, or submarginal design (see page 100)
2. Reflect subperiosteal flap; expose half the root and apical lesion (Fig 3-19)
3. Use tapered fissure bur to remove $\frac{1}{3}$ root apex; bevel apex faciolingually
4. Curette granulomatous tissue around apex; biopsy as needed; do not jeopardize adjacent tooth blood supply
5. Class I preparation about 3 to 4 mm into root canal using slow-speed micro-handpiece or ultrasonic tip.
6. Fill root end with stable, biocompatible, nonresorbable material

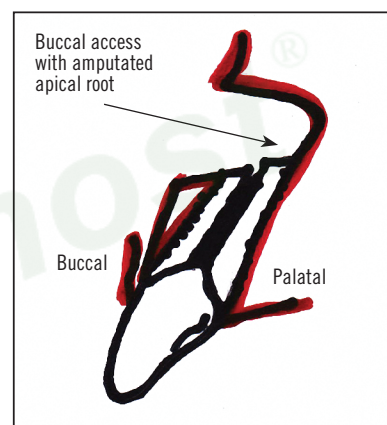


Fig 3-19 Apicoectomy. Note the angle at which the root is cut to improve access to the root canal (illustrated by Quoc Lu.)

Filling Material

- Amalgam; do not use if
 - Bloody field
 - < 3-mm-deep root end preparation (inadequate retention)
 - Limited access
- Mineral trioxide aggregate; does not need dry field but unknown long-term stability
- Composite resin; can be used in shallow, concave preparations (ie, molar roots) but needs dry field
- Reinforced zinc oxide cement

Miscellaneous Information

Average Suture Resorption Time

- Plain gut: 3 to 5 days
- Chromic gut: < 30 days but patient-dependent (enzymatic proteolysis)
- Polyglactin 9/10 and polyglycolic acid: 60 to 90 days (ester hydrolysis)
- Polydioxanone: Minimal resorption up to 90 days; then resorbed in 18 to 30 months (ester hydrolysis)

Distilled water

- Do not use for irrigation
- Hypotonic; enters cells due to osmotic gradient; can cause cell lysis/rapid cell death

Recommended Readings

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- The American Association of Oral and Maxillofacial Surgeons. AAOMS Task Force: White paper on third molar data. www.aaoms.org/docs/third_molar_white_paper.pdf. Accessed on 28 January 2015.
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Dental Implantology

Christopher Choi and Daniel Spagnoli

- ▶ Implant Components
- ▶ Presurgical Work-Up
- ▶ Osseointegration
- ▶ Surgical Principles
- ▶ Edentulous Patients
- ▶ Immediate Implants
- ▶ Implant Site Development
- ▶ Complications

Implant Components

Materials	Made of either Grade 4 commercially pure titanium (cpTi) or titanium alloy (Ti-6Al-4V) <ul style="list-style-type: none">• Ti-6Al-4V contains 6% aluminum and 4% vanadium and is stronger• An oxide layer is formed on the surface of the implant that is protective and allows osseointegration															
Size	Length (available from 6 to 16 mm) <ul style="list-style-type: none">• Longer implants increase surface area and bone-implant interface• Important for initial stability and resistance to rotational torque and shear forces Diameter (available from 3 to 6 mm) <ul style="list-style-type: none">• Wider implants increase surface area and bone-implant interface• Maximizing diameter (within appropriate constraints of alveolar bone)<ul style="list-style-type: none">– Minimizes interproximal space and potential food impaction and improves oral hygiene– Minimizes component fracture– Reduces incidence of screw loosening– Improves emergence profile of crown• Most stress on an implant is found at the crestal 5 mm; thus, width is more important than length in reducing stress on the implant															
Surface	Rough implant surface <ul style="list-style-type: none">• Surface roughness is categorized by Sa value (average height deviation in a given surface) <table><tr><th>Roughness</th><th>Sa value (μm)</th><th>Type of implant</th></tr><tr><td>Smooth</td><td>< 0.5</td><td>Machined implants Implant abutments</td></tr><tr><td>Minimally rough</td><td>0.5–1.0</td><td>3i implants</td></tr><tr><td>Moderately rough</td><td>1.0–2.0</td><td>Most modern implants</td></tr><tr><td>Severe roughness</td><td>> 2.0</td><td>Plasma-sprayed implants</td></tr></table> <p>Advantages</p> <ul style="list-style-type: none">• Increases surface area and bone-implant contact• Faster and stronger osseointegration compared to machined surface <p>Disadvantages</p> <ul style="list-style-type: none">• Increases plaque retention when exposed above bone, promoting peri-implantitis <p>Roughening techniques</p> <ul style="list-style-type: none">• Additive (titanium plasma spray)• Reduction (titanium oxide blasting, acid etching, laser etching, grit blasting)	Roughness	Sa value (μm)	Type of implant	Smooth	< 0.5	Machined implants Implant abutments	Minimally rough	0.5–1.0	3i implants	Moderately rough	1.0–2.0	Most modern implants	Severe roughness	> 2.0	Plasma-sprayed implants
Roughness	Sa value (μm)	Type of implant														
Smooth	< 0.5	Machined implants Implant abutments														
Minimally rough	0.5–1.0	3i implants														
Moderately rough	1.0–2.0	Most modern implants														
Severe roughness	> 2.0	Plasma-sprayed implants														
Implant design: Crest module	Crest module: Region extending from the implant body containing the abutment-implant connection (Fig 4-1) <ul style="list-style-type: none">• Connection to abutment: (> 20 designs) determines joint strength and lateral and rotational stability<ul style="list-style-type: none">– External<ul style="list-style-type: none">◦ Traditional design with butt-joint connection◦ Abutment screw loosening is problematic															

(Implant Components cont)

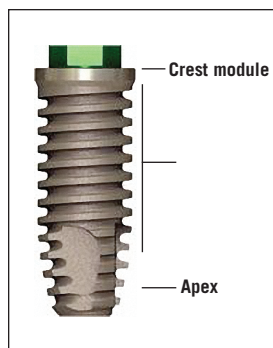


Fig 4-1 Crest module implant design. (Reprinted with permission from Misch CE. Dental Implant Prosthetics. St Louis: Elsevier, 2005:34.)

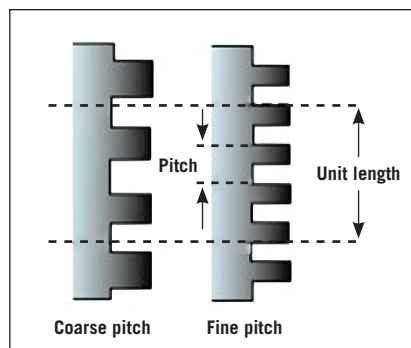


Fig 4-2 Coarse and fine pitch. (Reprinted with permission from Misch CE. Contemporary Implant Dentistry [ed 3]. St Louis: Elsevier, 2007:210.)

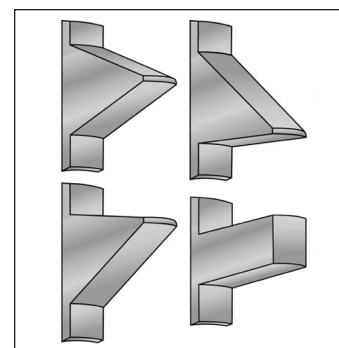


Fig 4-3 Different thread shapes. (Reprinted with permission from Misch CE. Contemporary Implant Dentistry [ed 3]. St Louis: Elsevier, 2007:210.)

	<ul style="list-style-type: none"> – Internal <ul style="list-style-type: none"> ◦ Design of most contemporary implants ◦ Incorporates an antirotational feature (hexagon, tripod, Morse taper, internal grooves) • Platform switching – Use of narrower restorative abutments on a wider implant body – Shown to decrease crestal bone resorption by preventing migration of epithelium past the implant-abutment interface, enhancing the connective tissue–osseous attachment in the crestal area
Implant design: Body	<ul style="list-style-type: none"> • Parallel walls <ul style="list-style-type: none"> – Larger surface area • Tapered walls <ul style="list-style-type: none"> – Allows compression of bone in poor-quality sites and distributes forces into surrounding bone – Facilitates placement into anatomically constricted sites (buccal concavities, adjacent teeth) • Antirotational hole or vent near apex is designed to resist torsional load • Parallel-wall versus tapered-wall implants <ul style="list-style-type: none"> – In sites with poor bone quality, underpreparation with tapered implant can achieve greater primary stability compared to that with parallel wall implant
Implant design: Thread geometry	<ul style="list-style-type: none"> • Pitch <ul style="list-style-type: none"> – Distance between adjacent threads (Fig 4-2) – Smaller pitch = more threads on implant = greater surface area per unit length • Shape (Fig 4-3) <ul style="list-style-type: none"> – Square: less stress in compressive and shear forces – V shape – Buttress thread – Reverse buttress • Depth <ul style="list-style-type: none"> – Deeper threads increase surface area and are better suited for soft bone – Shallow threads allow easier placement of implant in hard bone

Presurgical Work-Up

Medical history	Risk factors for implant failure <ul style="list-style-type: none"> • Periodontal disease • Smoking • Prior radiation treatment • Diabetes
Dental history	Patients with parafunctional habits (bruxism, clenching) may require <ul style="list-style-type: none"> • Additional implants to reduce overload • Modification of anterior teeth to re-create appropriate incisal guidance to avoid posterior interferences during excursions • Night guard to transfer force off implant restoration
Restorative space requirements (Fig 4-4)	<ul style="list-style-type: none"> • Implant position is restoration driven and dictated by the final position of the tooth • Vertical space for fixed restoration (from crestal bone to occlusal plane) <ul style="list-style-type: none"> – Minimum: 8 mm (cemented), 6 mm (screw-retained) <ul style="list-style-type: none"> ◦ 1 mm occlusal metal restoration (2 mm for porcelain) ◦ 5 mm abutment for cement restoration (1 mm subgingival margin, total 6 mm) ◦ 2 mm soft tissue attachment – Ideal: 9 to 10 mm in posterior, 10 to 12 mm in anterior – If > 12 mm, teeth will be elongated and may require addition of pink tones in esthetic regions
Surgical space requirements	Implants should be placed <ul style="list-style-type: none"> • 1.5 mm from adjacent tooth to prevent excess interproximal bone loss • 3 mm from adjacent implant to prevent excess interproximal bone loss • With 1 mm of buccal/lingual bone • 3 mm apical to gingival margin for appropriate emergence profile of crown • 2 mm palatal to buccal wall in esthetic zone for appropriate crown emergence and to prevent buccal bone loss • 2 mm above inferior alveolar nerve • 5 mm anterior to mental foramen to avoid anterior loop of the mental nerve
Bone quality (Fig 4-5)	Types of bone <ol style="list-style-type: none"> 1: Predominantly cortical bone (anterior mandible) <ul style="list-style-type: none"> – Overheating potential – Tapping of bone recommended to facilitate implant placement 2: Thick cortical bone and dense cancellous bone (mandible, anterior maxilla) 3: Thin cortical bone and dense cancellous bone (maxilla) 4: Predominantly cancellous bone (posterior maxilla) <ul style="list-style-type: none"> – Poor bone quality leads to lower success – Consider osteotome technique to compress denser bone laterally next to implant – Consider underpreparation of site <ul style="list-style-type: none"> • Best bone for implant placement is types 2 and 3 (dense, cancellous bone)

(Presurgical Work-Up cont)

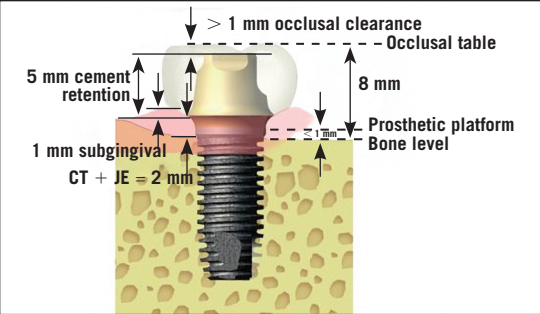


Fig 4-4 Space requirements for fixed restoration. CT, connective tissue; JE, junctional epithelium. (Reprinted with permission from Misch CE. Dental Implant Prosthetics. St Louis: Elsevier, 2005:166.)

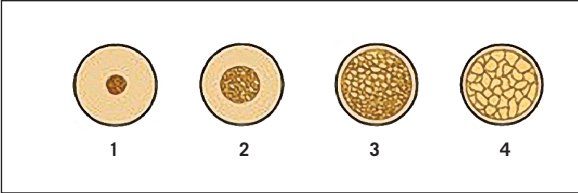


Fig 4-5 Lekholm and Zarb classification of bone, types 1 to 4. (Adapted with permission from Lekholm U, Zarb GA. Patient selection and preparation. In: Brånemark P-I, Zarb GA, Albrektsson T (eds). Tissue-Integrated Prostheses. Chicago: Quintessence, 1985:202.)

Gingival biotype

- Assessment of thickness can be made by **visibility of periodontal probe** through gingival margin.
 - Thick** tissue: Probe is **not visible**
 - Thin** tissue: Probe is **visible**
- Thick gingival biotype is associated with greater soft tissue stability, more predictable healing, less gingival discoloration from titanium show, and less gingival recession.

Imaging

Imaging technique	Radiation dose (µSv)	Comments
Periapical radiograph	5.0	<ul style="list-style-type: none"> Useful for single implant cases Helpful for intraoperative verification of implant position <ul style="list-style-type: none"> Best for implant-to-bone visualization without scatter Limitations: Image magnification, 2D
Panoramic radiograph	3.0–24.3	<ul style="list-style-type: none"> Useful for multiple implants, identification of vital structures Limitations: Distortion, image magnification, 2D
CBCT: Small FOV	11–674	<ul style="list-style-type: none"> Radiation doses vary tremendously between machines Accurate, 3D view of anatomy, bone quantity/quality
CBCT: Large FOV	30–1,073	<ul style="list-style-type: none"> Can manufacture surgical guides Expensive, highest levels of radiation

2D, two-dimensional; CBCT, cone beam computed tomography; FOV, field of view; 3D, three-dimensional.

Osseointegration

- **Definition:** Direct **structural** and **functional connection** between **bone** and the surface of an **implant** that can survive normal loading conditions
- Timing for osseointegration was traditionally **3 months in mandible** and **6 months in maxilla**; improvements in implant surfaces have decreased this window of time
- Implant stability is characterized by both mechanical and biologic stability (Fig 4-6)
- **Mechanical** stability is initially high immediately after surgery and declines over time
- **Biologic** stability eventually takes over as new bone forms on the implant surface and osseointegration occurs
- The weakest time for implant stability is **2 to 4 weeks** after placement
- Peri-implant endosseous healing is described by the following
 - **Osteoconduction:** Recruitment of osteogenic cells to the implant surface
 - **Contact osteogenesis:** De novo bone formation on the implant surface
 - **Distance osteogenesis:** New bone formation on the walls of the osteotomy site toward the implant surface
- **Implant success** definition: absence of implant mobility, peri-implant radiolucency, and symptoms (pain, infection, numbness); crestal bone loss < 0.2 mm per year following first year of function; restorability

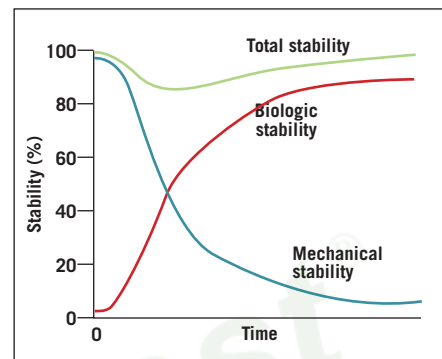


Fig 4-6 Stability of dental implants over time. (Reprinted with permission from Bedrossian E. *Implant Treatment Planning for the Edentulous Patient: A Graftless Approach to Immediate Loading*. St Louis: Mosby, 2011:12.)

Surgical Principles

Stage 1: Surgical Placement of Implant

- Position is guided by use of a surgical stent or “free-handed” with previously established parameters
- Implant should be angulated so that it emerges in the center of posterior teeth and at the cingulum of anterior teeth
- Implant site preparation requires copious irrigation, sharp drills, and **speeds less than 2,000 rpm** to prevent overheating of bone
 - Thermal necrosis occurs at **47°C**
- Flap design
 - A **2-mm** cuff of **keratinized** tissue should be maintained around implants to better withstand functional stresses, improve hygiene, and avoid complications of mobile tissue, including chronic inflammation, irritation, and peri-implantitis
 - Midcrestal incision with minimal mucoperiosteal elevation to visualize alveolar crest
 - Papilla-sparing incision: Avoids reflection of papilla to prevent gingival recession
 - Flapless surgery
 - Maintains periosteal blood supply, potentially minimizing crestal bone loss
 - Minimizes gingival recession and helps preserve dental papilla in the esthetic zone
 - Requires presence of adequate keratinized tissue
 - Measure thickness of soft tissue with periodontal probe to place implant at proper depth

Stage 2: Surgical Uncovering of Implant

- In areas of abundant keratinized tissue, implants can be uncovered using **punch biopsy** instruments or a **laser**
- Type of flap used depends on amount of keratinized tissue on buccal aspect of implant(s)
 - > 5 mm: Midcrestal incision is made in keratinized tissue, with or without resective contouring, and interimplant sutures are placed (Fig 4-7a)
 - 4 to 5 mm: Reverse cutback incision with pedicles rotated into interimplant space (Fig 4-7b)
 - 3 to 4 mm: Lateral flap advancement with distal release; attached tissues distal to implant site are repositioned mesially (Fig 4-7c)

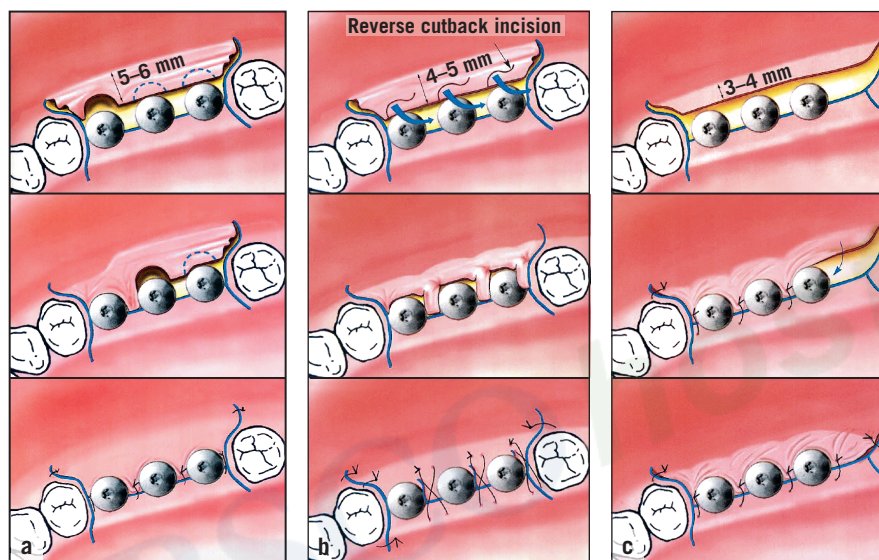


Fig 4-7 (a) Midcrestal incision flap. (b) Reverse cutback incision flap. (c) Lateral flap advancement. (Reprinted with permission from Sclar AG. *Soft Tissue and Esthetic Considerations in Implant Therapy*. Chicago: Quintessence, 2003.)

Single-Stage (Nonsubmerged) Versus Two-Stage (Submerged) Approach

- Traditional protocol called for two-stage approach in which implant is submerged initially and uncovered at a second stage
- In single-stage or nonsubmerged approach, a healing abutment or provisional prosthesis is immediately placed on the implant (see page 129)
 - Avoids need for second uncovering procedure
 - Requires adequate **primary stability** (> 35 Ncm)
 - Develops and maintains soft tissue architecture in esthetic zone

Edentulous Patients

Implant reconstruction of the edentulous patient is influenced by a number of considerations. The restorative plan needs to be established with considerations regarding availability of bone, length of treatment, cost, and patient ability to maintain hygiene.

Prosthetic Options

Implant-supported fixed prosthesis

- **Fixed porcelain-fused-to-metal (PFM) bridge** – Requires adequate bone (**tooth-only defect**) to avoid unesthetic crown lengths – Requires **6 to 8 implants** with **precise positioning**
 - Location of implants
 - Primary site: First molar, important for occlusal force (2 implants)
 - Secondary site: Canine, important for canine guidance (2 implants)
 - Tertiary site: Lateral incisor or second premolar (2 to 4 implants)
 - **Advantages:** Most esthetic and natural option
 - **Disadvantages:** Most expensive and technique-sensitive option; may require bone grafting
- **Hybrid prosthesis** (fixed-detachable prosthesis, profile prosthesis)
 - Denture teeth connected to a metal framework with acrylic resin
 - Replaces teeth and bone in patients with resorption
 - Vertical space required for prosthesis is **15 mm** from **soft tissue** to the planned occlusal plane; more space allows for thickening of prosthesis, more strength, and less likelihood of fracture
 - 2 mm: space under bar for hygiene
 - 8 mm: cast framework
 - 2 mm: acrylic
 - 3 mm: teeth
 - Alveolar reduction may be required
 - To achieve appropriate vertical distance
 - To hide transition line of prosthesis if alveolar ridge is visible on smile line
 - **Cantilever** length depends on **anteroposterior (AP) spread** (distance between anterior and posterior implants measured from midline of arch) and number of implants
 - **Less than 1.5 times the AP spread** with 4 implants
 - Implants should be maximally spaced to lengthen the AP spread and cantilever length in order to achieve first molar restoration
 - Anatomy of arch determines AP spread
 - Square-shaped arches, least AP spread
 - V-shaped arches, most AP spread
 - **Advantages:** Avoids bone grafting in atrophic maxilla; can load implants immediately
 - **Disadvantages:** Prosthesis can be bulky and affect speech; requires removal by dentist at regular intervals for hygiene; costly
 - Number of implants required depends on availability of bone in radiographic zones (Fig 4-8)

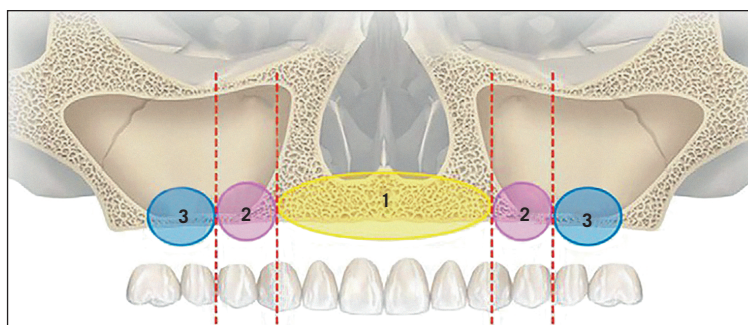


Fig 4-8 Radiographic zones used to determine extent of maxillary atrophy. (Reprinted with permission from Bedrossian E. Rescue implant concept: The expanded use of the zygoma implant in the graftless solutions. Oral Maxillofacial Surg Clin North Am 2011;23:257–276.)

Adequate bone present (10 mm)	Surgical approach	Prosthetic options
Zones 1, 2, 3	Traditional implants	Fixed PFM bridge
Zones 1, 2	Angled implants (all-on-four)	Hybrid
Zone 1	Zygomatic implants	Hybrid
Insufficient bone in any zone	Quad zygoma	Hybrid

Implant-retained, tissue-supported prosthesis

- **Overdenture**

- Mandible: 2 implants 20 mm apart (or as far apart as possible with respect to mental foramina) with individual Locator (Dentsply) abutments or splinted with a rigid interconnecting bar
- Maxilla: 4 implants connected with a bar
 - Highest failure rate among implant prostheses due to poorer quality bone, prosthetic design, higher loads
- Vertical space requirement is a minimum of **12 mm** from **soft tissue**
 - 1 mm for below bar for hygiene
 - 3 mm for bar and Hader clip (or 5 mm for bar and O-ring)
 - 8 mm for tooth
- **Advantages:** Cheapest option; dramatically improves stability of mandibular dentures
- **Disadvantages:** Maxillary overdenture has high failure rate

Combination syndrome: Atrophy of the edentulous anterior maxilla that develops in patients with a partially edentulous mandible and preserved anterior mandibular teeth. Treatment includes placing implants in the posterior maxilla to distribute forces to posterior occlusion and off the anterior maxilla.

For the **atrophic maxilla**, the surgical approach and subsequent implant reconstruction (without bone grafting) can be determined based on the extent of atrophy in **three radiographic zones** (see Fig 4-8).

All-on-four

- 4 implants are placed in a configuration to **maximize AP spread**
 - 2 anterior implants
 - 2 posterior implants placed in zone 2 and **angled** from the crest anterior to the apex to avoid the pneumatized maxillary sinuses or the mental nerves
- Biomechanically, 4 implants are sufficient to support a fixed, implant-borne prosthesis (hybrid only) because the anterior- and posterior-most implants bear the entire load, even when more implants are placed in between
- All-on-six : Large arch form, larger ridge discrepancies, or in situations of higher potential failure (maxilla, smoking, diabetes, periodontal disease, bruxism)

Zygomatic implant

- Available lengths: 35.0 to 52.5 mm
- Used with a hybrid prosthesis only
- Full-arch restoration also requires a **minimum of 2 (preferably 4) implants in the anterior maxilla**
- Traditional or intrasinus technique
 - Path is from **premolar region** through the **maxillary sinus** and entering the midportion of the **zygomatic body** (use care to avoid penetration into the orbit or pterygomaxillary space; Fig 4-9a)
 - A window is created in the sinus for **direct visualization** of the implant pathway
 - The sinus membrane should be removed or reflected so that it does not attach to the implant
 - Emergence of the implant is palatal to the palatal cusp of the maxillary premolar (Fig 4-9b)
 - Palatal emergence of implant head results in a bulky prosthesis and challenges in speech and hygiene
- Zygoma anatomy-guided approach (ZAGA) (Fig 4-10)
 - The implant head is positioned in a more vertical position over the crest of the alveolar ridge to allow for an improved prosthesis
 - In cases of pronounced buccal concavity of the lateral maxillary sinus wall, the implant body can be “extrasinus”

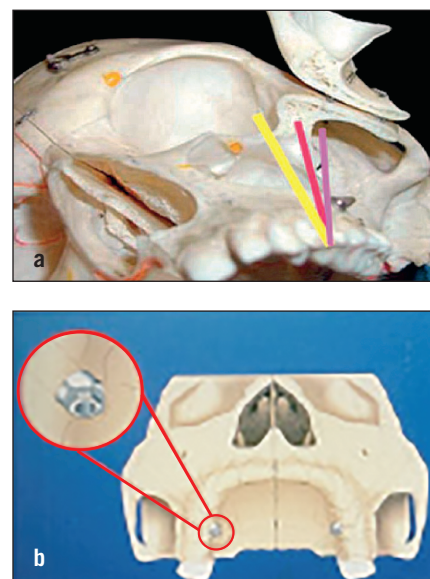


Fig 4-9 (a) Placement of zygomatic implant in the midportion of the zygomatic body (*red lines*). Avoid penetration into the orbit or pterygomaxillary space. (b) Emergence of the zygomatic implant. (Reprinted with permission from Bedrossian E. *Implant Treatment Planning for the Edentulous Patient: A Graftless Approach to Immediate Loading*. St Louis: Mosby, 2011:64.)

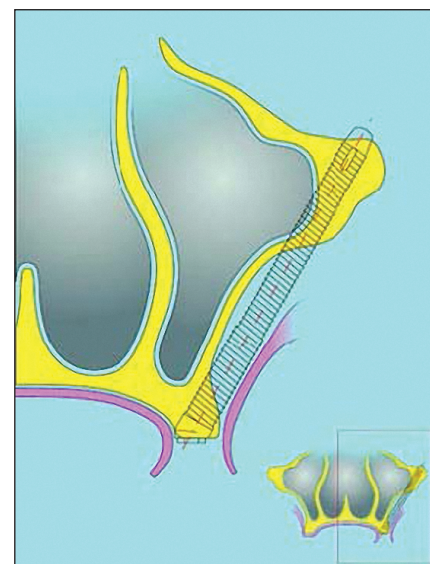


Fig 4-10 Extrasinus technique. (Reprinted with permission from Aparicio C, Manresa C, Francisco K, et al. Zygomatic implants placed using zygomatic anatomy-guided approach versus the classical technique: A proposed system to report rhinosinusitis diagnosis. *Clin Implant Dent Relat Res* 2014;16:627–642.)

Immediate Implants

- Should not be performed if proper angulation of implant cannot be attained to meet restorative requirements
- Requires **2 to 3 mm** of **apical** bone for primary stabilization
- For anterior teeth, implants should be placed in palatal bone of extraction socket, leaving a 2- to 3-mm gap
- Bone grafting should be done to fill the gap between the implant and the bony walls

Immediate Temporization of Implant

- Provisional crown can help mold gingival contours and improve esthetics during soft tissue healing
- Single-unit provisional crown should be left in **infraocclusion**
 - Multiple implants should be splinted together
- **Screw-retained** provisional crown is preferred as cement can migrate into the gingival crevice and cause peri-implantitis and failure
- **Primary stability** is mandatory
 - Requires an insertion torque of at least **30 to 35 Ncm**
 - Facilitated by implant design with deep threads and with a fine pitch
- Methods for single crown
 - Preoperative laboratory preparation of abutment and provisional crown from an implant analog placed in the dental cast
 - Chairside abutment preparation and fabrication of provisional crown by relining a hollow shell
 - Transfer impression of implant for laboratory fabrication of provisional crown to be delivered later that day or the following day

Implant Site Development

Soft Tissue Augmentation

Epithelialized palatal graft (free gingival graft) (Fig 4-11)

- Indication: To increase width of **keratinized** tissue around implants
- Timing: At nonsubmerged implant placement, stage-two uncovering surgery, or after definitive restoration
- Harvest site: **Full-thickness** graft from smooth area of palate in the premolar/molar area
- Recipient site: **Split-thickness flap**; graft is secured to periosteal bed
- Allografts (acellular dermal allograft) can be used to avoid donor site surgery but has **greater shrinkage** rate
- Palatal stent helps alleviate postoperative discomfort

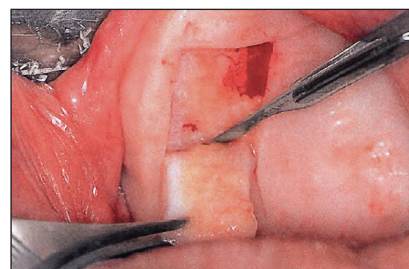


Fig 4-11 Epithelialized palatal graft. (Reprinted with permission from Sclar A. Soft Tissue and Esthetic Considerations in Implant Dentistry. Chicago: Quintessence, 2003:129.).

Subepithelial connective tissue graft (Fig 4-12)

- Indications: To **thicken** gingival tissue (can achieve up to **3 mm**) in order to prevent recession and subsequent metal show as well as to improve soft tissue contour
- Timing: Prior to stage-two uncovering surgery or nonsubmerged implant placement
- Harvest site: In premolar area 2 to 3 mm apical to the gingival margin; an incision is made on the palate, and a subepithelial incision is then made parallel to the external surface to harvest connective tissue
- Recipient site: A suprapariosteal **pouch** is created, and the graft is placed inside and secured with sutures

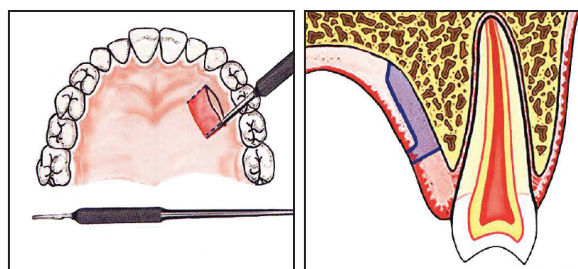


Fig 4-12 Subepithelial connective tissue graft. (Reprinted with permission from Sclar A. Soft Tissue and Esthetic Considerations in Implant Dentistry. Chicago: Quintessence, 2003:129.)

Palatal roll technique (Fig 4-13)

- Indications: To enhance soft tissue contours and thicken tissue
- Timing: At stage-two uncovering surgery
- Harvest and recipient sites: Subepithelial connective tissue pedicle from palate is elevated, rolled, and secured under the buccal flap

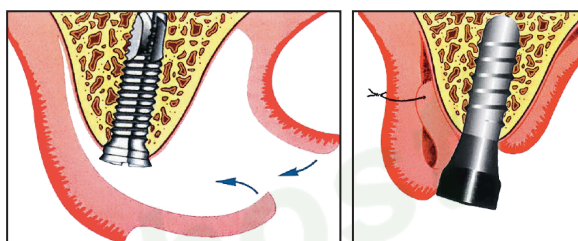


Fig 4-13 Palatal roll technique. (Reprinted with permission from Sclar A. Soft Tissue and Esthetic Considerations in Implant Dentistry. Chicago: Quintessence, 2003:119.)

Pedicle flap from palate (vascularized interpositional periosteal connective tissue flap) (Fig 4-14)

- Random-pattern periosteal-connective tissue flap from the palate is rotated into the recipient site and positioned beneath the recipient-site flap
- Indications: **Simultaneous hard and soft tissue site development** in the maxillary anterior for **correction of large-volume defects**

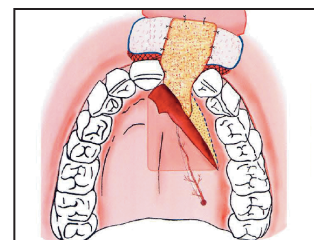


Fig 4-14 Pedicle flap from palate. (Reprinted with permission from Sclar A. Soft Tissue and Esthetic Considerations in Implant Dentistry. Chicago: Quintessence, 2003:172.)

Bone Grafting

Definitions

- **Osteoinduction:** Growth factors stimulate mesenchymal cells to differentiate into osteoblastic lineages
- **Osteoconduction:** Acts as a matrix for bone growth
- **Osteogenesis:** Transplanted osteoblasts and periosteum produce own bone

Materials

Autogenous bone

- Gold standard
- Osteogenic, osteoinductive, and osteoconductive
- Sites
 - Corticocancellous: Tibia, iliac crest
 - Cortical: Cranium, mandibular ramus, mandibular symphysis

Allograft (human), xenograft (animal)

- **Osteoconductive**
- Processed graft material available in blocks or particles of different size
- Xenografts resorb slower than allografts

Tissue engineering

- Classic tissue engineering **triangle** is composed of
 - **Source** of cells
 - **Signal**
 - **Scaffold**
 - Recombinant human bone morphogenetic protein-2 (rhBMP-2) and acellular collagen sponge (ACS)
 - **Osteoinductive**
 - Member of **transforming growth factor** (TGF) superfamily
 - US Food and Drug Administration cleared for alveolar ridge augmentation for defects associated with extraction sockets and sinus elevation bone grafting
 - Incubation time on ACS carrier: 15 minutes; should be used within 2 hours after wetting
 - Allow 4 to 6 months of healing and/or bone formation (depending on size of site) prior to implant placement

Ridge Augmentation

	Width	Height	Comments
Onlay	X		Fixation of cortical graft with screws; potential for resorption
Ridge split	X		Minimum 3 mm of ridge width required
Mesh	X	X	Mesh exposure can occur (mandible > maxilla), and removal is tedious
Sandwich osteotomy	Some	X	Interpositional graft with plating of superior segment
Distraction osteogenesis	X		Latency: 5 to 7 days Distraction: 0.5 mm twice a day (1 mm per day) Consolidation: 2 to 3 months

Socket Preservation

- The natural evolution after an extraction is one of **bone loss**, most significantly affecting the **buccolin-gual** dimension (50% loss in 6 months)
- Socket healing resembles **intramembranous** bone formation
 - **Clot** formation and stabilization of fibrin network, days 1 to 3
 - **Granulation** tissue near crest and **provisional matrix**, day 7
 - **Woven** bone and closure of socket by keratinized epithelium, month 1
 - Mature **lamellar** bone, month 2

- Bone **remodeling**: Turnover of bone characterized by **osteoblastic** formation balanced with **osteoclastic** resorption
- Bone **modeling**: Adaptive change in bone size and shape in response to presence or absence of external mechanical forces
- Extraction sockets with **wall defects** or **thin buccal** bone benefit from bone grafting
- Techniques
 - Placement of allograft/xenograft (0.5 to 1.0 mL of 250- to 1,000- μ m-sized particles)
 - Barrier **membranes** can be used for buccal wall defects and act to mechanically exclude invasion by nonosteogenic cell populations from surrounding soft tissues (**guided bone regeneration**)
 - rhBMP-2/ACS alone or mixed with corticocancellous particles is placed into sockets
 - De novo bone formation

Sinus Elevation

Anatomy

- With loss of posterior teeth, the sinus cavity expands (**pneumatization**), and crestal bone resorbs
- Maxillary sinus cavity volume for the average adult is **14.75 mL** (range between 9.5 and 20 mL)
- **Sinus dimension**: Width = 2.5 cm; height = 3.75 cm; depth = 3 cm
- Sinus membrane lines the maxillary sinus cavity
 - Thickness varies from 0.13 to 0.50 mm
 - Ciliated epithelium functions to drain sinus contents superiorly through an ostium into the **middle meatus** of the nose

Two Techniques

Lateral wall approach: Creation of lateral window and elevation of the maxillary sinus membrane

- Direct visualization of sinus cavity
- Can graft bone in larger areas
- For simultaneous implant placement, adequate alveolar bone is required to obtain primary stability of implant (**3 to 5 mm**)
- Sinus **perforation** is most common complication
 - Small perforations: Sinus membrane folds over itself and covers hole
 - Larger perforations: Cover with a collagen membrane
 - Very large perforations: Abandon grafting and reenter in 6 months

Internal elevation (summer technique)

- Use of osteotomes through extraction socket to elevate floor of sinus \leq **2 mm**
- Does not allow visualization of the maxillary sinus membrane
- Typically performed in conjunction with implant placement

Technique used depends on available height of alveolar bone

Height	Procedure
< 3 mm	Sinus membrane elevation with lateral approach; wait 4 to 6 months before implant placement
3–8 mm	Sinus membrane elevation with lateral approach and simultaneous implant placement
8–10 mm	Internal sinus membrane elevation with implant placement

Complications

Most common surgical complications	<ul style="list-style-type: none"> • Hemorrhage-related (24%): Ecchymosis, hematoma <ul style="list-style-type: none"> – Implant site osteotomy in mandibular canine area can injure a perforator vessel and cause swelling of the floor of the mouth and potential airway compromise • Neurosensory disturbance (7%) • Mandible fracture (0.3%)
Implant failure	<ul style="list-style-type: none"> • Risk factors (failure rate) <ul style="list-style-type: none"> – Radiation therapy (maxilla 25%, mandible 6%) – Maxillary overdentures (19%) – Type 4 bone (16%) – Smoking (11%) – Implants shorter than 10 mm (10%) – Diabetic patients (9%)
Prosthetic complications	<ul style="list-style-type: none"> • More common with implant prosthesis <ul style="list-style-type: none"> – Overdenture clip/attachment loosening (30%) – Resin veneer fracture on fixed partial dentures (22%) – Overdenture requiring relining (19%) – Overdenture clip/attachment fracture (17%) • Abutment screw loosening (8%) caused by excursive forces <ul style="list-style-type: none"> – Single crowns > multiple-unit fixed prosthesis > overdentures
Peri-implantitis	<ul style="list-style-type: none"> • Present in 5% to 10% of osseointegrated implants • Mucosal inflammation with loss of supporting bone • Most commonly occurs with implant-supported overdentures • Normal implant probing depths ≤ 5 mm • Normal bone loss is ≤ 1.5 mm at year 1 and < 0.2 mm per year thereafter <ul style="list-style-type: none"> – Causes <ul style="list-style-type: none"> ◦ Excess cement ◦ Mechanical factors: Occlusal overload, poor prosthetic design, parafunctional habits ◦ Biologic factors: Bacteria similar to periodontitis: gram-negative anaerobic bacteria (<i>Aggregatibacter actinomycetemcomitans</i>, <i>Porphyromonas gingivalis</i>, <i>Prevotella intermedia</i>) ◦ Patient-related factors: Smoking, systemic disease, periodontitis ◦ Surgery-related factors: Overheating bone, poor primary stability, dehiscence of thin bone • Management: Challenging and unpredictable – Remove etiology (eliminate overload, control infection) <ul style="list-style-type: none"> – Surgical exposure and debridement <ul style="list-style-type: none"> ◦ Remove granulation tissue with instruments that do not scratch titanium surface (titanium or plastic scalers) – Surface decontamination <ul style="list-style-type: none"> ◦ Remove bacteria biofilm ◦ Agents: Saline, abrasive pumice, citric acid, chlorhexidine, hydrogen peroxide, tetracycline, lasers – Guided bone regeneration <ul style="list-style-type: none"> ◦ Fill osseous defect and eliminate probing depth

Recommended Readings

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Orthognathic Surgery

David Alfi and Jaime Gateno

- ▶ Treatment Planning
- ▶ Maxillary Surgery
- ▶ Mandibular Ramus Osteotomies
- ▶ Genioplasty
- ▶ Stability of Orthognathic Surgery
- ▶ Controversies in Orthognathic Surgery
- ▶ Obstructive Sleep Apnea Syndrome

Indications

Orthognathic surgery is indicated when

- A patient has a jaw deformity—size, position, orientation, shape, or symmetry
- The jaw deformity cannot be camouflaged with orthodontics either because the occlusal discrepancy is beyond the limits of orthodontic treatment or because it is significant despite a good occlusion (eg, hemifacial microsomia, where a patient may have severe jaw deformities and normal occlusion)
- The deformity is causing an impairment or is comorbid with other conditions

Impairments	Common comorbidities
<ul style="list-style-type: none">• Appearance• Function<ul style="list-style-type: none">– Mastication– Speech– Breathing– Socialization	<ul style="list-style-type: none">• TMJ disease• Obstructive sleep apnea• Myofascial pain
TMJ, temporomandibular joint.	

Treatment Planning

Clinical Work-Up for Orthognathic Surgery

Formal treatment planning is needed twice: before orthodontics (presurgical plan) and before surgery (surgical plan). A work-up collects information and processes it to formulate a diagnosis and a treatment plan. Any work-up includes a thorough medical history and a physical examination—including facial measurements. An orthognathic work-up also includes diagnostic items.

A **presurgical** work-up requires the following items

- Clinical photographs
- Dental models
- Cephalograms
- Panoramic radiograph

A **surgical** work-up can be done using two different routines

- Traditional planning
- Virtual planning

Surgical work-up

Traditional planning	Virtual planning
<ul style="list-style-type: none"> • Clinical photographs • Dental models mounted on a semi-adjustable articulator • Cephalograms and prediction tracings • Panoramic radiograph 	<ul style="list-style-type: none"> • Clinical photographs • Digital dental models • Computed tomography (CT) scan correctly oriented to an anatomical frame of reference

Clinical photographs: Standardized intraoral and facial photos. The facial photos, taken in natural head position (NHP), should include profile and frontal views with the lips relaxed and when smiling.

Cephalogram: Standardized plain radiograph of the face and cranium. It should include lateral and frontal views, and the lateral cephalogram should be taken with the patient's lips relaxed and with the mandible in centric relation (Fig 5-1).

Dental models: Three-dimensional models of the teeth. They can be made of stone (casts) or be digital and are used to evaluate the dentition and to establish a new occlusion.

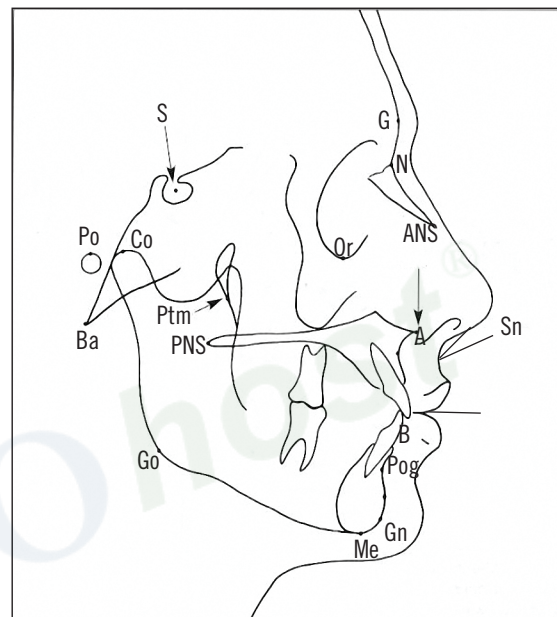


Fig 5-1 Cephalometric landmarks. A, A-point; ANS, anterior nasal spine; B, B-point; Ba, basion; Co, condylion; G, glabella; Gn, gnathion; Go, gonion; Me, menton; N, nasion; Or, orbitale; PNS, posterior nasal spine; Po, porion; Pog, pogonion; Ptm, pterygomaxillare; S, sella; Sn, subnasale; St, stomion.

Facial Examination

An examination of the face is performed with the patient in NHP, in which the patient is standing or sitting erect with the face looking forward toward the horizon. The NHP places the patient into a posture that is most natural to an observer. A facial examination should be done systematically, one region at a time.

Upper third

- Area from the hairline (trichion) to glabella
- Assess eyebrow shape, position, and symmetry
- Male eyebrows are larger, more horizontal, and level with the supraorbital rims
- Female eyebrows slope upward, peaking about 10 mm above the supraorbital rims
- The superior orbital rims should project about 10 mm in front of the cornea

Middle third

- Area from the glabella to base of the nose (subnasale); includes the eyes, nose, and cheeks
- The lateral orbital rims should be 8 to 12 mm behind the cornea
- The intercanthal distance approximates alar base width
- Lower nose projection is affected by the anteroposterior (AP) position of the maxilla
- The malar eminence is usually located 10 to 15 mm lateral to and 15 to 20 mm inferior to the lateral canthus
- The nasolabial angle is normally 100 degrees \pm 10 degrees; it is greater in females than in males

Lower third

- Area from base of the nose (subnasale) to bottom of the chin (menton)
- Evaluate the lips statically and dynamically for symmetry
- Assess maxillary vertical position by measuring the maxillary lip at rest incisal show, normally ranging from 0.5 to 5 mm; it varies according to
 - Age: Decreases with age, where 5 mm can be considered normal in an adolescent, 0.5 mm is normal in a 60-year-old
 - Sex: Females show more tooth than males
 - Ethnicity: Whites have greater incisal show than Asians; Asians have more incisal show than blacks
 - Upper lip length: The longer the upper lip at rest, the less the incisal show, and vice versa
 - Incisal attrition: The greater the attrition, the less the incisal show
- Lip width, the distance from one oral commissure to the other, approximates interpupillary distance
- The upper lip occupies 30% of the height of the lower facial third
- The lower lip should have 25% more vermilion than the upper lip
- The bigonial width should be 30% less than the bizygomatic width
- Neck: Chin-throat angle is normally ~110 degrees

Cephalometric Analysis

Assess AP maxillary position

- Steiner analysis uses the SNA angle (Fig 5-2)
 - When this analysis was developed, it was believed that the cranial base of all individuals was alike; however, now we know that there is wide variation in the inclination of the anterior cranial base (SN); and thus, the SNA angle should be interpreted with caution
- Ricketts analysis uses maxillary depth
 1. Draw the NA line (line that crosses nasion [N] and A-point [A])
 2. Draw Frankfort horizontal (FH) (line that crosses the upper margin of the bony external auditory canals [porion] and the lowest point of the infraorbital rim [orbitale])
 3. Measure the angle at the intersection of these lines
- McNamara measures the **distance from A to N perpendicular** (Fig 5-3)
 1. Draw N perpendicular (a line that crosses N and is perpendicular to FH)
 2. Measure the distance from A to N perpendicular
 3. If A is in front of N perpendicular, the number is positive; if it is behind, the number is negative

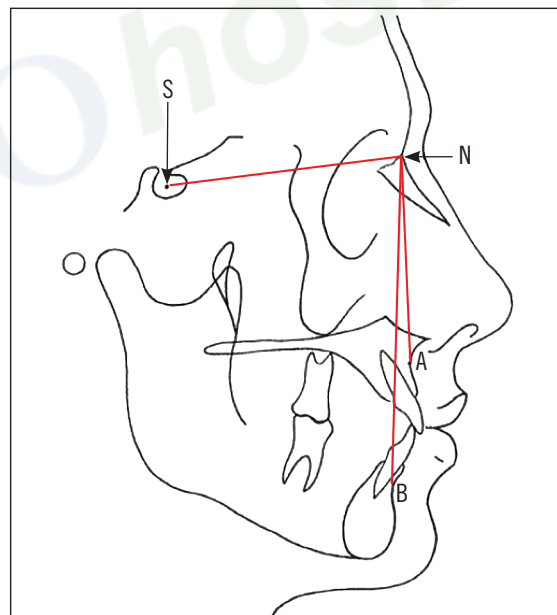


Fig 5-2 Steiner analysis. A, A-point; B, B-point; N, nasion; S, sella.

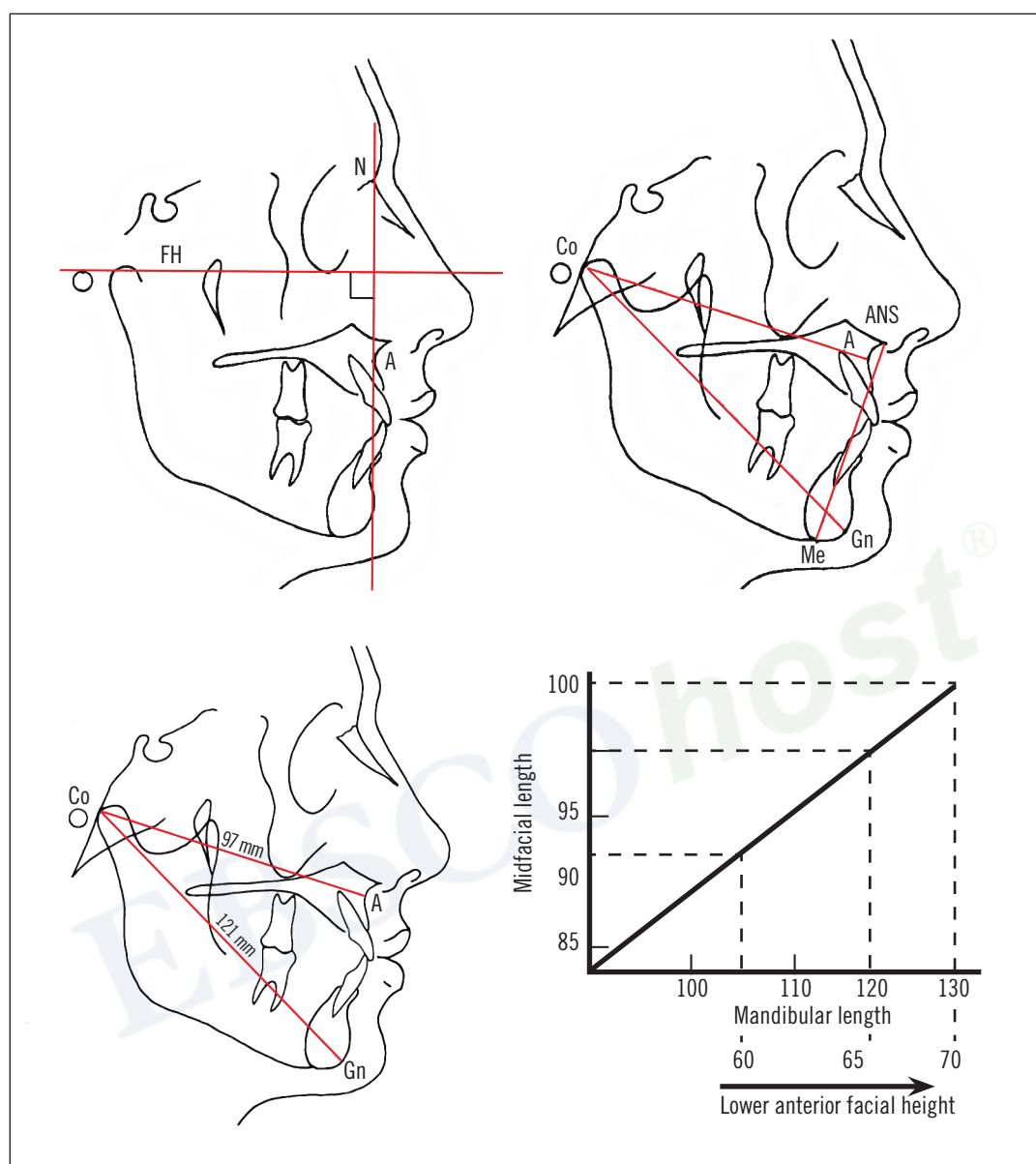


Fig 5-3 McNamara analysis. A, A-point; ANS, anterior nasal spine; Co, condylion; Gn, gnathion; Me, menton; N, nasion.

			AP maxillary position		
Analysis	Measurement	Ideal	Retrognathia	Normal	Prognathism
Steiner	SNA	81 ± 3 degrees	< 79 degrees	79 to 84 degrees	> 84 degrees
Ricketts	Maxillary depth	90 ± 4 degrees	< 86 degrees	86 to 94 degrees	> 94 degrees
McNamara	Distance from A to N perpendicular	0–1 mm	< 0 mm	0 to 1 mm	> 1 mm

Assess AP mandibular position

- Steiner analysis uses the SNB angle
 - As with the SNA angle, this measurement should be interpreted with caution because of possible distortion caused by an anterior cranial base that is either too steep or too flat
- Downs analysis uses the **facial angle** (Fig 5-4)
 - Draw the facial plane as a line that crosses N and pogonion (Pog)
 - Measure the angle at the intersection of the facial plane and FH
- McNamara analysis measures the distance from **Pog to N perpendicular**
 - Draw N perpendicular, the line that crosses N and is perpendicular to FH
 - Measure the distance from Pog to N perpendicular
 - If Pog is in front of N perpendicular, the number is positive; if it is behind, it is negative

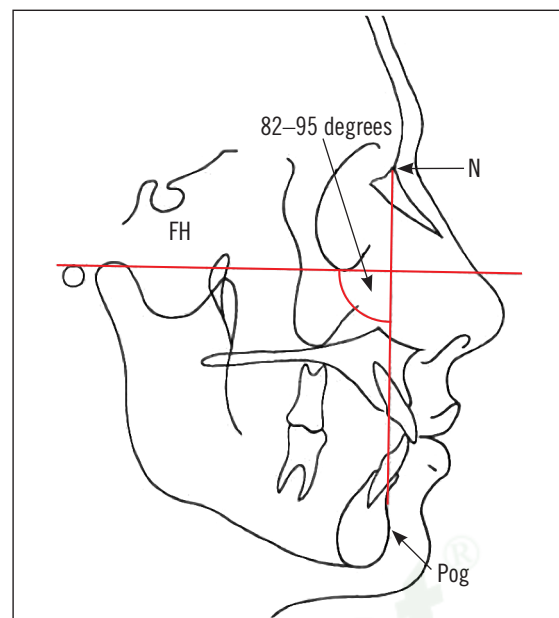


Fig 5-4 Downs analysis.

			AP maxillary position		
Analysis	Measurement	Ideal	Retrognathia	Normal	Prognathism
Steiner	SNB	79 ± 3 degrees	< 76 degrees	76 to 82 degrees	> 82 degrees
Downs	Facial angle	90 ± 4 degrees	< 86 degrees	86 to 94 degrees	> 94 degrees
McNamara	Distance from Pog to N perpendicular	Mixed dentition -8 to -6 mm	< -8 mm	-8 to -6 mm	> -6 mm
		Adult female -4 to 0 mm	< -4 mm	-4 to 0 mm	> 0 mm
		Adult male -2 to +2 mm	< -2 mm	-2 to +2 mm	$> +2$ mm

Assess the AP discrepancy between the maxilla and the mandible

- Steiner analysis uses the **ANB angle**, which measures difference in AP position between one jaw and the other; A and B-point (B) represent the apical bases of the maxilla and mandible, respectively
- Wits appraisal measures position of one jaw with respect to the other (apical bases)
 - Draw the occlusal plane as a line that crosses the tips of the mandibular first molar and premolars
 - Project A on the occlusal plane: Draw a line that crosses A and is perpendicular to the occlusal plane; find the point where the perpendicular line crosses the occlusal plane, and call this point AO
 - Do the same for B, and call this point BO
 - Measure the distance from BO to AO: If BO is behind AO, give the distance a negative sign; if BO is in front, give the distance a positive sign

- McNamara analysis measures the **maxillomandibular discrepancy**, which is the size discrepancy between the maxilla and mandible
 - Measure the midfacial length, the distance from condylion (Co) to A
 - Measure the mandibular length, the distance from Co to Pog
 - Calculate the maxillomandibular discrepancy by subtracting the midfacial length from the mandibular length

			AP maxillomandibular discrepancy		
Analysis	Measurement	Ideal	Skeletal Class II	Normal	Skeletal Class III
Steiner	ANB	2 ± 2 degrees	> 4 degrees	4 to 0 degrees	< 0 degrees
Wits	Wits appraisal	Males: -1.0 mm	> 2.6 mm	2.6 to -5 mm	< -5 mm
		Females: 0 mm	> 3.5 mm	3.5 to -3.7 mm	< -3.7 mm
McNamara	Maxillo-mandibular discrepancy	Mixed dentition 19–21 mm	< 19 mm	19 to 21 mm	> 21 mm
		Adult women 25–27 mm	< 25 mm	25 to 27 mm	> 27 mm
		Adult men 30–33 mm	< 30 mm	30 to 33 mm	> 33 mm

Assess maxillary incisor inclination

- Steiner **maxillary incisor inclination analysis**
 - Draw the long axis of the maxillary incisor
 - Draw the NA line, a line than crosses N and A
 - Measure the (superior) angle were the lines cross

			Inclination of the maxillary incisors		
Analysis	Measurement	Ideal	Retroclined	Normal	Proclined
Steiner	Maxillary incisor inclination	22 ± 6 degrees	< 16 degrees	16 to 28 degrees	> 28 degrees

Assess mandibular incisor inclination

- Steiner mandibular incisor inclination
 - Draw the long axis of the mandibular incisor
 - Draw the NB line, a line than crosses N and B
 - Measure the (inferior) angle were the lines cross
- Incisor mandibular plane angle (IMPA) of Downs and Tweed
 - Draw the long axis of the mandibular incisor
 - Draw the mandibular plane as a line
 - Measure the (superior) angle were the lines cross

			Inclination of the mandibular incisors		
Analysis	Measurement	Ideal	Retroclined	Normal	Proclined
Steiner	Lower incisor inclination	25 ± 7 degrees	< 18 degrees	18 to 32 degrees	> 32 degrees
Downs	IMPA	90 ± 5 degrees	< 85 degrees	85 to 95 degrees	> 95 degrees

Assess facial type

- Steiner mandibular plane angle analysis
 - Draw the line SN
 - Draw the mandibular plane
 - Measure the angle at the intersection of the mandibular plane and SN
- Frankfort mandibular plane angle (FMA) of Downs and Tweed
 - Measure the angle at the intersection of the mandibular plane and FH

			Facial type (shape)		
Analysis	Measurement	Ideal	Short face*	Normal	Long face [†]
Steiner	Mandibular plane angle	32 ± 2 degrees	< 30 degrees	30 to 34 degrees	> 34 degrees
Downs/Tweed	FMA	24 ± 3 degrees	< 21 degrees	21 to 27 degrees	> 27 degrees

*Long ramus, acute gonial angle, horizontal growth, over-closed mandible, decreased lower facial height.

[†]Short ramus, obtuse gonial angle, vertical growth, mandible is rotated open, increased lower facial height.

Assess the chin

- Holdaway ratio
 - Draw a line that crosses N and B (NB)
 - Measure the distance from NB to the incisal edge of the mandibular incisor and the distance from NB to Pog
 - Calculate the ratio of the distances (NB to incisal edge of mandibular incisor:NB to Pog)
- The ideal ratio for white males is 1:1; for white females it is 0.5:1

Growth Evaluation

Growth cessation can be determined by

- Serial physical examinations
- Serial cephalograms
- A hand-wrist film
- Looking at the maturation of the cervical vertebrae on a lateral cephalometric radiograph (Fig 5-5)
 - There are six stages of cervical vertebral maturation
 - Mandibular and craniofacial growth peak along with stature in stages 3 and 4
 - As cervical vertebrae mature, they develop a concavity on the inferior border and assume a more rectangular shape (*arrows*)

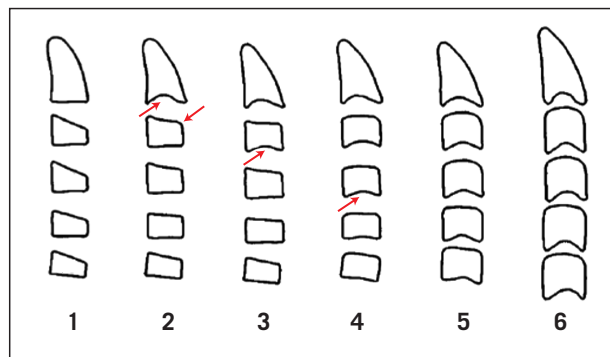


Fig 5-5 Cervical spine evaluation.

Presurgical Orthodontics

The goals of presurgical orthodontics are to normalize and coordinate the dental arches.

- A dental arch is normalized when
 - All the teeth are aligned (orderly arrangement in an arch); the teeth are not displaced, tipped, or rotated
 - The teeth are leveled; they are vertically even—there are no steps in the incisal edges or marginal ridges
 - The curve of Spee is flat or minimal
 - The lingual cusps of the mandibular posterior teeth are 1 mm below the buccal cusps
 - The palatal cusps of the maxillary posterior teeth are 1 mm below the buccal cusps
 - All interdental spaces are closed unless they are needed to manage a tooth-size discrepancy
 - All dental compensations have been removed
- The dental arches are coordinated when
 - The maxillary and mandibular dental arches have the same shape
 - The maxillary and mandibular dental arches have corresponding sizes
 - The tooth sizes of the maxillary and mandibular teeth correspond—they have the correct size ratio
- Compensations are nature's attempt at camouflaging a jaw deformity
 - In a Class III malocclusion, the maxillary incisors are inclined labially; the mandibular incisors are inclined lingually
 - In a Class II malocclusion, the maxillary incisors can be tipped labially (division 1) or lingually (division 2); the mandibular incisors are tipped labially
 - In patients with a Class II deep bite malocclusion with a deep curve of Spee, it may be advantageous to do surgery before the curve of Spee is leveled; this prevents intrusion of the mandibular incisors by orthodontic mechanics and foreshortening of the lower face
- Presurgical orthodontics may worsen a malocclusion
- Before surgery
 - Archwires should be passive
 - Rectangular orthodontic wires should be fully engaged in the slots
 - Surgical hooks (soldered or crimped) should be in place to facilitate maxillomandibular fixation (MMF)

Maxillary Surgery

Le Fort I Osteotomy

Indications	Correction of deformities that affect the size, position, orientation, and shape of the maxilla
Incision	3–5 mm above the mucogingival junction from zygomatic buttress to zygomatic buttress
Soft tissue dissection (subperiosteal)	<p>Intraorally expose</p> <ul style="list-style-type: none"> • Piriform aperture • Infraorbital foramina • Zygomatic buttresses • Pterygomaxillary fissure <p>Intranasally expose</p> <ul style="list-style-type: none"> • Nasal floor • Caudal septum • Lower aspect of lateral nasal wall
Osteotomy	<p>Horizontal osteotomy from piriform rim to tuberosity at least 5 mm above the apices of teeth</p> <p>Lateral nasal wall osteotomy</p> <ul style="list-style-type: none"> • From piriform rim to just in front of the descending palatine artery (DPA) • Average distance from piriform rim to DPA = 34 mm <p>Septal separation</p> <ul style="list-style-type: none"> • From anterior nasal spine to posterior nasal spine; avoid excessive force; fracture propagation to the cribriform plate can cause anosmia <p>Pterygomaxillary separation</p> <ul style="list-style-type: none"> • The average height of the pterygomaxillary suture is 14.6 mm • Place a finger on the palatal aspect of the tuberosity • Cut from lateral to medial and from top to bottom (away for the vessels) • Avoid excessive force <ul style="list-style-type: none"> – Use very sharp osteotome – Several passes with narrow osteotome (eg, 5 mm) delivers less force than a single pass with a wide osteotome (eg, 15 mm) • Some surgeons prefer to cut through the tuberosity rather than the pterygomaxillary suture
Downfracture	<ul style="list-style-type: none"> • As a rule, use finger pressure • If resistance is met, revise all cuts • Rarely, instruments (spreaders or disimpaction forceps) may be needed
Mobilization	<ul style="list-style-type: none"> • Push down in tuberosity area until fracture is complete • Rock maxilla side to side • To advance, push maxilla forward from the tuberosities

(Le Fort I Osteotomy cont)

Vessel management	Preservation of DPA is not mandatory but advisable; studies show good outcomes with ligation, yet the DPAs provide good blood supply to the maxilla and contribute to flap nourishment
Segmentation	<p>Changes the shape and width of the maxilla</p> <p>Interdental cuts</p> <ul style="list-style-type: none"> • Can be made between the lateral incisors (2s) and the canines (3s) or between the canines (3s) and first premolars (4s) • Advantages of cuts between 2s and 3s – Allows change in axial inclination of anterior teeth – Facilitates management of tooth-size discrepancy (eg, narrow maxillary lateral incisors) <ul style="list-style-type: none"> – No need to open interradicular spaces orthodontically as there is naturally enough space for the cut – Less risk of iatrogenic root injury: Cutting between two uni-radicular teeth (2s and 3s) is less dangerous than cutting between one uni-radicular and one bi-radicular tooth (3s and 4s) • Advantages of cuts between 3s and 4s: Larger anterior segment; more blood supply <p>Palatal cuts</p> <ul style="list-style-type: none"> • Avoid the midline; make the cut(s) parasagittally where the bone is thinner and the soft tissue thicker
Fixation	4 miniplates (1.5-mm screws): 2 plates at the piriform buttresses; 2 plates at the zygomatic buttresses
Closure	<ul style="list-style-type: none"> • Alar cinch suture: Prevents widening of the alar base • V-Y advancement: Prevents loss of visible vermillion

Surgically Assisted Rapid Palatal Expansion (SARPE)

- Basically, a 2-piece Le Fort I osteotomy without downfracture
- Correction of severe (>10 mm) transverse deficiency after the midpalatal suture is closed for correction of isolated transverse deficiencies

Technique

- A fine osteotome is placed anteriorly in the midpalatal suture and tapped backward until the suture opens
- An expander device is installed before or after the osteotomies; it can be tooth borne or bone borne
- No archwire should be present
- Activation of expander begins 5–7 days after surgery at 0.5 mm/day (one turn [0.25 mm] twice a day)
- Overexpand, as relapse is common and significant

Le Fort I Osteotomy Complications

Intraoperative	<p>Bleeding</p> <ul style="list-style-type: none">• Possible sources are –<ul style="list-style-type: none">– Pterygoid venous plexus –– Descending palatine artery<ul style="list-style-type: none">◦ Descends through the pterygopalatine canal, emerging from the greater palatine foramen◦ A Le Fort I osteotomy breaks the pterygopalatine canal horizontally, placing the vessel at risk◦ An overt injury results in brisk bleeding that must be controlled with ligation◦ An occult injury can result in delay bleeding (see below for details)– Sphenopalatine artery– Internal maxillary artery– Posterosuperior alveolar artery• Can be minimized with the use of – Reverse Trendelenburg position (10 degrees)<ul style="list-style-type: none">– Local vasoconstrictors<ul style="list-style-type: none">◦ Injection of 1:100,000 epinephrine around the wound◦ Topical application of cocaine or oxymetazoline on nasal mucosa– Hypotension <p>Damage to teeth</p> <p>Unwanted fracture</p> <ul style="list-style-type: none">• Horizontal fracture of pterygoid process can leave the end of the process and medial pterygoid muscle attached to the maxilla and limit maxillary advancement; if this occurs, separate the pterygoid process from the tuberosity• Propagation of fracture to the septum and cribriform plate• Propagation of fracture to skull base <p>Anosmia</p> <ul style="list-style-type: none">• Caused by cribriform plate fracture <p>Blindness</p> <ul style="list-style-type: none">• Very rare (9 published cases)• May be caused by<ul style="list-style-type: none">– Base of skull fracture– Hypoperfusion of the optic nerve– Arterial aneurysm
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(Le Fort I Osteotomy Complications cont)

<p>Postoperative</p>	<p>Malocclusion</p> <p>Infection</p> <ul style="list-style-type: none"> • Data supports the use of prophylactic antibiotics to decrease risk of infection <p>Bleeding</p> <ul style="list-style-type: none"> • Immediate postoperative period – Most common cause is “silk deficiency,” one or more vessels were not ligated, cauterized, or welded at surgery <ul style="list-style-type: none"> – Undiagnosed bleeding diathesis • Late postoperative period <ul style="list-style-type: none"> – Clot lysis around occult vessel injury – Rupture of false aneurysm or arteriovenous fistula <p>Hardware failure</p> <p>Delayed healing and nonunion</p> <ul style="list-style-type: none"> • Rare; risks include <ul style="list-style-type: none"> – Inferior maxillary repositioning – Large movements – Bruxism – Unoperated mandible – Hardware failure – Poor blood supply – Heavy elastics – Occlusal interferences • Management – For delayed healing consider <ul style="list-style-type: none"> ◦ Occlusal equilibration ◦ Discontinuation of heavy elastics ◦ Non-chewy diet ◦ MMF – For nonunion: Surgical revision, with removal of fibrous union, re-application of rigid fixation, and bone grafting <p>Avascular necrosis</p> <ul style="list-style-type: none"> • Rare (less than 1%); risk factors include <ul style="list-style-type: none"> – Smoking – Vascular disease (eg, atherosclerosis) – Thrombophilia – Cleft palate – Previous surgery – Large surgical movements; especially, AP and transverse – Maxillary segmentation – Long operating time – Laceration of palatal tissues – Impingement on palatal tissues from segmental osteotomies – Impingement on mucosa by splint • Can result in pulp necrosis, periodontal defects, gingival recession, bone resorption, and segmental or complete loss of maxilla • Treatment depends on the extent and severity of the necrosis and includes debridement and reconstruction; hyperbaric oxygen therapy has been used to halt the progression of necrosis and establish early improvement
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Mandibular Ramus Osteotomies

	Sagittal split osteotomy (SSO)	Vertical ramus osteotomy (VRO)	Inverted-L osteotomy
Indications	Correction of deformities that affect the size, position, orientation, and shape of the mandible	Correction of mandibular prognathism	Correction of severe deformities that affect the size, position, orientation, and shape of the mandible
Advantages	<ul style="list-style-type: none"> No MMF is required Occlusion can be checked intraoperatively and immediately postoperatively No bone graft is usually required Can be used to correct most mandibular deformities: retrognathism, micrognathia, prognathism, macrognathia, open bite, malrotation, and distortion 	<ul style="list-style-type: none"> Inferior alveolar nerve (IAN) injury is rare Not associated with iatrogenic TMJ disc displacement or condylar degeneration 	<ul style="list-style-type: none"> Allows for large translations and rotations No MMF is required Occlusion can be checked intraoperatively and immediately postoperatively
Disadvantages	<ul style="list-style-type: none"> Moderate risk of IAN disturbance Unfeasible when large movements are needed <ul style="list-style-type: none"> Very large advancements (> 15 mm) Very large counterclockwise rotations Big yaw or roll rotations—needed to correct severe asymmetries—that move the proximal segment out of alignment Can produce iatrogenic 	<ul style="list-style-type: none"> Requires MMF Possible condylar sag Occlusion cannot be checked intraoperatively Postoperatively, occlusion can only be checked after the bone has healed Cannot be used to correct retrognathia, micrognathia, or anterior open bite 	<ul style="list-style-type: none"> Bone graft is required when used for advancement Advancement requires external approach <ul style="list-style-type: none"> Scar Longer surgery Possible facial nerve injury

Surgical Techniques

	SSO	VRO	Inverted-L osteotomy
Anatomy	<ul style="list-style-type: none"> Vertically, the IAN is closest to the teeth in the third molar region; lowest below the first molar Transversely, the IAN is closest to the buccal cortex in the third molar region; farthest from it at the level of the first molar Placing the body cut between the first and second molar minimizes the risk of IAN injury 	<ul style="list-style-type: none"> Masseteric artery passes 8 mm above the sigmoid notch Internal maxillary artery is posterior and medial to the condylar neck The antilingula has been used to locate the lingula, yet a cadaveric study found the antilingula posterior to the lingula in 45% of specimens The mandibular foramen is rarely less than 7 mm away from the posterior border 	<ul style="list-style-type: none"> Marginal mandibular branch of the facial nerve at risk with extraoral approach <ul style="list-style-type: none"> Multiple branches in 81% of individuals Deep to the platysma, within the superficial layer of the deep cervical facial (investing layer) Lateral to facial vein – Cadaveric studies find it up to 1.2 cm below the inferior border of the mandible, yet in patients whose heads are rotated and hyperextended, the nerve usually dips lower
Incision	Intraoral	Intraoral or extraoral (neck)	Intraoral or extraoral (neck)
Soft tissue dissection	<ul style="list-style-type: none"> Lateral: Expose ramus and body Inferior: Expose inferior border between the first and second molars (for body cut) Medial: Expose medial ramus above the lingula 	<p>Intraoral</p> <ul style="list-style-type: none"> Lateral: Expose entire ramus Inferior: Expose entire inferior border of ramus to prevent iatrogenic injury to facial vessels Medial: No dissection before the osteotomy; after osteotomy, mobilize the proximal segment laterally, and minimally dissect the lateral pterygoid muscle to prevent condylar sagging <p>Extraoral</p> <ul style="list-style-type: none"> Retromandibular approach Risdon approach Submandibular approach 	<p>Intraoral</p> <ul style="list-style-type: none"> Same as VRO <p>Extraoral</p> <ul style="list-style-type: none"> Risdon approach Submandibular approach

(Surgical Techniques cont)

	SSO	VRO	Inverted-L osteotomy
Osteotomy	Cuts <ul style="list-style-type: none"> • Medial: Horizontal cut of medial cortex of ramus just above lingula; extend horizontally behind it • Sagittal: Monocortical osteotomy extending from anterior extent of medial osteotomy to interproximal space between the first and second molars (Dalpont modification) • Body: A vertical monocortical osteotomy from the anterior extent of the sagittal cut to the inferior border of the mandible, where it should be bicortical • Separation proceeds from anterior to posterior; may expose the IAN, which should stay medially 	Cut <ul style="list-style-type: none"> • Vertically oriented cut from sigmoid notch to the angle of the mandible, behind the mandibular foramen 	Cuts <ul style="list-style-type: none"> • Horizontal: Above the lingula; from the anterior border of the ramus to a point just behind the mandibular foramen • Vertical: From the posterior limit of the horizontal cut to the inferior border of the mandible, in front of the angle
Fixation	<ul style="list-style-type: none"> • Proximal segments must be in centric relation • The inferior borders of the proximal and distal segments should be aligned • Three options for rigid fixation, from biomechanically best to worst <ul style="list-style-type: none"> – Three positional (bicortical) screws placed in an inverted L configuration: two up, one down – Hybrid: one to two positional screws plus a plate with monocortical screws 	<ul style="list-style-type: none"> • MMF for 6 weeks • Some use rigid fixation but is technically difficult intraorally 	Usually fixed with a reconstruction plate
Pediatric considerations	<ul style="list-style-type: none"> • The lingula and the mandibular foramen are more superior and posterior • There is an increased risk of greenstick fracture • The greater amount of cancellous bone in the ramus can result in a sagittal split that propagates to the posterior border to the mandible rather than fracturing lingually behind the nerve • The developing third molars are very small and can be removed after the osteotomy is completed 		

Complications

	SSO	VRO	Inverted-L osteotomy
Intraoperative	<ul style="list-style-type: none">• Bleeding: Possible sources are buccal vessels, bone marrow, inferior alveolar vessels, retro-mandibular vein, facial vessels• IAN injury: At 20 months, a third of patients still have sensory disturbance• Bad split (unfavorable fracture)<ul style="list-style-type: none">– Most commonly because of incomplete inferior border cut– Also associated with presence of third molars; should be removed 9 to 12 months prior to surgery– If it occurs, (1) complete the split as originally intended; (2) repair the fracture with rigid fixation, reestablishing the proximal and distal segments; (3) position the proximal and distal segments as planned and fix the osteotomy– If the repair is tenuous, use postoperative MMF; if the reduction is poor, postpone any additional procedures• Changes in intercondylar width—widening or narrowing—are usually caused by lag screws when they are used to close a transverse interfragmentary gap and displace the proximal	<ul style="list-style-type: none">• Bleeding<ul style="list-style-type: none">– IAN– Masseteric artery– Facial artery• IAN injury (rare)• Misdirected osteotomy<ul style="list-style-type: none">– Occurs when the osteotomy ends at the posterior border of the mandible rather than at the inferior border– Increases the risk of condylar sagging and resultant malocclusion• Medial displacement of the proximal segments: If it occurs, look for the proximal segment at the condylar neck,	<ul style="list-style-type: none">• Bleeding (rare)• Nerve injury<ul style="list-style-type: none">– IAN– Cervical and mandibular branches of facial nerve when a neck incision is used• Proximal segment fracture: Usually at the corner of the L

(Complications cont)

	SSO	VRO	Inverted-L osteotomy
Postoperative	<ul style="list-style-type: none"> • Infection • Bleeding • Immediate malocclusion – <ul style="list-style-type: none"> Condylar malposition – Mechanical deformation (warping) caused by bendable fixation and/or large mechanical loads (eg, large advancements) • Late malocclusion <ul style="list-style-type: none"> – Hardware failure and nonunion – Occlusal interferences caused by postoperative orthodontics – Dental relapse – Condylar resorption; more prevalent in females, patients with steep mandibular planes, and patients with open bite • TMJ disc disorder or osteoarthritis • Hardware failure • Nonunion 	<ul style="list-style-type: none"> • Infection • Bleeding • Immediate malocclusion: <ul style="list-style-type: none"> Caused by condylar malposition • Late malocclusion <ul style="list-style-type: none"> – Occlusal interferences caused by postoperative 	<ul style="list-style-type: none"> • Infection • Bleeding • Immediate malocclusion <ul style="list-style-type: none"> – Condylar malposition – Mechanical deformation (warping) caused by bendable fixation and/or large mechanical loads (eg, large movements) • Late malocclusion <ul style="list-style-type: none"> – Hardware failure and nonunion – Occlusal interferences caused by postoperative orthodontics – Dental relapse – Condylar resorption • Hardware failure • Nonunion

Genioplasty

Indications	<ul style="list-style-type: none"> • Microgenia • Chin asymmetry • Macrogenia: If at all possible, avoid moving the chin backward; removing support to the genial soft tissues can result in chin ptosis; instead, camouflage macrogenia with clockwise rotation of both jaws
Incision	<ul style="list-style-type: none"> • Labial sulcus incision from first premolar to first premolar, > 3 mm away from the mucogingival line • Warning: Branches of the mental nerve can run superficially, just under the mucosa

(Genioplasty cont)

Soft tissue dissection	<ul style="list-style-type: none"> • Cut through the mentalis muscles, leaving muscle stumps on the bone, which are needed to resuspend the muscles • Differentiate the fibers of the mentalis muscles from the orbicularis; mentalis fibers run vertically, orbicularis fibers run horizontally • Deglove the chin • Find mental foramina and mental nerve • Dissect the nerves proximally for 1 to 2 cm to prevent neuropraxia
Osteotomy	<ul style="list-style-type: none"> • ≥ 5 mm below apices of teeth • ≥ 5 mm away from the mental foramina; the IAN loops in front of the mental foramina
Fixation	Options <ul style="list-style-type: none"> • Chin plate: Use 2.0-mm bicortical screws • Screws • Wires
Closure	<ul style="list-style-type: none"> • Carefully reapproximate the mentalis muscles with a slowly resorbing suture • Failure of mentalis approximation can result in chin ptosis, ie, witch's chin

Stability of Orthognathic Surgery

During the first postsurgical year, treatment of Class II problems is more stable than Class III problems. In the first postsurgical year the following movements can be placed in three categories of hierarchy of stability:

- Highly stable
 - Maxillary impaction
 - Mandibular advancement
 - Genioplasty (any direction)
- Stable
 - Maxillary advancement
 - Maxillary impaction and mandibular advancement
 - Maxillary advancement and mandibular setback
- Unstable
 - Isolated mandibular setback
 - Maxillary inferior repositioning
 - Widening of the maxilla; greater relapse in the molar region
- Long-term instability
 - Bony and dental changes seen after the first postsurgical year are not because of surgery but are the result of growth, adaptive dental movements, or pathologic conditions (eg, condylar resorption)
 - Changes > 2 mm are more commonly seen in Class II than in Class III patients

Controversies in Orthognathic Surgery

Orthognathic Surgery in Growing Patients

Skeletal Class II	Skeletal Class III
If the deformity is impairing the airway or the patient's psychosocial development, consider early surgery, because a hypoplastic mandible has limited growth potential and risk for relapse is low	<ul style="list-style-type: none"> • Early surgery will most likely result in relapse • Recommend waiting for growth completion unless significant psychosocial problems exist and the patient is willing to accept a second operation

Double Jaw Surgery: Maxillary or Mandibular Surgery First

Maxilla first	Mandible first
<ul style="list-style-type: none"> • When the mandibular osteotomy is a VRO • When the mandibular deformity leaves doubt in the ability to achieve stable fixation; a bad split may make rigid fixation of the mandible impossible 	<ul style="list-style-type: none"> • Mandible not in centric relation (CR): In certain patients (eg, severe micrognathia, severe condylar erosion, muscular dystonia), it is difficult to place the mandible in CR for the planning records; in this scenario, doing mandibular surgery first avoids maxillary malposition because of erroneous CR recording • Maxillary surgery moves the mandible past the point of pure condylar rotation: This occurs when the occlusal plane is severely canted or when the occlusal plane is very steep; current planning methods cannot duplicate condylar translation; thus, in these cases, it is best to do mandibular surgery first • Maxillary bone is very thin: Propping the mouth open during mandibular surgery can overload the maxilla, resulting in loss of fixation

Management Options for Idiopathic Condylar Resorption

	Orthognathic surgery	Arthroplasty with prosthetic replacement	Arthroplasty with rib autograft
Advantages	<ul style="list-style-type: none"> • Simpler surgery, less morbidity • If successful, no need for future surgeries 	No relapse	If successful, no need for additional surgeries
Disadvantages	Possible relapse, even if previously stable	<ul style="list-style-type: none"> • More extensive operation than orthognathic surgery; may have CN VII weakness, neck scar, etc • May need revision about every 15 years but may last longer; an issue in young patients since no growth potential 	<ul style="list-style-type: none"> • Donor site morbidity • More extensive operation than orthognathic surgery • Possible overgrowth

Obstructive Sleep Apnea Syndrome

Definition

Obstructive sleep apnea syndrome (OSAS) is characterized by repetitive collapse of the upper airway during sleep. These episodes of airway collapse produce a collection of physiologic derangements.

- Sleep fragmentation
- Hypoxemia
- Hypercapnia
- Marked swings in intrathoracic pressure
- Impaired cognition

Prevalence: 2% to 4% of the population.

Symptoms

- Nocturnal
 - Loud snoring
 - Witnessed breathing interruptions
 - Awakenings due to gasping or choking
 - Nocturia
- Diurnal
 - Waking up unrefreshed
 - Morning headaches
 - Daytime sleepiness: Should be quantified using the Epworth Sleepiness Scale; scores range from 0 to 24; normal ranges from 0 to 8
 - Impaired concentration and memory
- Criteria for OSAS diagnosis
 - Five or more obstructive events per hour of sleep AND presence of symptoms
 - Fifteen or more obstructive events per hour of sleep, irrespective of symptoms

Sleep Testing: Polysomnography

Obstructive events

- Apnea: Breathing interruption > 10 seconds
- Hypopnea: More than 50% decrease in nasal airflow or more than 2/3 decrease in tidal volume with 3% decrease in oxygen saturation
- Respiratory effort–related arousals (RERAs): More than 50% decrease in nasal pressure and increased work of breathing associated with arousal

Indexes that report the frequency of obstructive events

- Apnea hypopnea index (AHI): Apneas and hypopneas per hour
- Respiratory disturbance index (RDI): Apneas, hypopneas, and RERAs per hour

Indexes that report severity of hypoxia

- Oxygen saturation nadir
- Total time of hypoxia

OSAS Severity

	RDI	AHI
Mild	10–30	5–15
Moderate	30–50	15–30
Severe	> 50	> 30

Cephalometry

Recommended when surgery is being considered.

- Evaluate facial skeleton and cranium
- Evaluate airway (at minimum, 3 measurements)
 - Soft palate length
 - Distance from posterior nasal spine to uvula tip
 - Mean: 35 mm
 - Posterior airway space
 - Smallest anteroposterior distance between the base of the tongue and the posterior pharyngeal wall
 - Mean: 11 mm
 - Hyoid to mandibular plane distance
 - From the anterosuperior limit of the hyoid to the mandibular plane
 - Ideal less than 15 mm
 - This distance is proportional to height of the pharynx

Sleep Endoscopy

- Not yet a standard test
- Done in the operating room with full anesthesia support
- Patients lay prone and receive propofol to induce sleep; nasopharyngoscopy is performed while the patient is sleeping
- Proponents believe that the findings help them stratify surgery patients into phase I or phase II treatment; they reason that
 - Patients with limited airway obstruction will do well after phase I surgeries
 - Those with complete collapse are better off skipping phase I, moving to maxillomandibular advancement

Fujita Classification

- Classification of anatomical sites of obstruction in the upper airway
 - Type I: Narrow oropharynx (retropalatal); large tonsils, uvula, and pillar webbing
 - Type II: Oral and hypopharyngeal obstruction (retropalatal and retrolingual); low arched palate and large tongue
 - Type III: Hypopharyngeal obstruction (retrolingual only); retrognathia, floppy epiglottis, enlarged lingual tonsils
- Most OSA patients have combined problems
- 20% have discrete type II-related OSA
- 10% have discrete type I-related OSA

Treatment

Medical

- Behavior modification
 - Positional therapy (eg, avoid supine position)
 - Weight loss
 - Exercise
 - Avoidance of alcohol and sedatives
- Continuous positive airway pressure (CPAP)
- Oral appliances
 - For all users, success rate is 47%
 - Better outcomes in patients with
 - Smaller body mass index (BMI)
 - Mild to moderate OSAS
 - Supine OSAS
 - Better tolerated than CPAP
 - Can produce jaw pain and/or malocclusion

Surgical

- For OSAS caused by morbid obesity that is refractory to weight management, consider **bariatric surgery**
- **Nasal surgery:** It does not improve OSAS but can increase tolerance to CPAP; it may help those patients that need high CPAPs because of nasal obstruction (septal deviation, turbinate hypertrophy)
- **Tonsillectomy and/or adenoidectomy:** For patients with severe adenotonsillar hyperplasia, a common pediatric condition
- **Orthognathic surgery:** For patients with severe jaw deformities, ie, maxillary and mandibular retrognathia
- For all other patients that are not morbidly obese, have no severe jaw deformity, and have no significant tonsillar hyperplasia, there are two options
 - Phase I: Multilevel surgery
 - Phase II: Maxillomandibular advancement
- For patients that failed medical therapy and other surgical therapies, consider tracheostomy; also a good alternative for patients that cannot tolerate more invasive procedures; it is 100% curative

Phase I multilevel surgery

Combination of different procedures, each aimed at increasing the size of the pharyngeal airway at a specific location.

Phase I procedures	Operation	Effect
Uvulopalatopharyngoplasty (UPPP) or W-palatoplasty	Shortens the soft palate and removes the tonsils	Increases oropharyngeal airway
Base of tongue reduction (radiofrequency, endoscopic robotic)	Removes tissue from base of tongue	Increases retroglossal airway
Hyoid suspension	Suspends the hyoid to the bony chin; brings hyoid forward	Increases retroglossal airway
Genioglossus advancement	Advances the bony segment that contains the attachments of the genial muscles (genioglossus and geniohyoid)	Increases tension on genioglossus muscles, preventing upper airway collapse

- Cure rate of phase I multilevel surgeries is 13%—defined as postoperative AHI < 5
- The success of phase I multilevel surgeries is 55%—defined as postoperative AHI < 20 AND a \geq 50% reduction in AHI

Maxillomandibular advancement (MMA)

- With the exception of a tracheostomy, MMA is the most effective operation for the treatment of OSAS in adults
- Using an endpoint of an AHI < 5, the cure rate is reported as 43.2%
- Using surgical success defined as postoperative AHI < 20 and a \geq 50% reduction in AHI, the success rate of this operation has been estimated to be 86.0%
- The rate of major complications is 1% and the minor complication rate is 3.1%; the major complications are mostly cardiac events, and the majority of minor complications are malocclusion (44%) and sensory disturbances of the face (14%)

Recommended Readings

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Trauma

Daniel E. Perez and Edward Ellis III

- ▶ Clinical Evaluation
- ▶ Anesthesia in the Traumatized Patient
- ▶ Principles of Fixation
- ▶ Soft Tissue Injuries
- ▶ Dentoalveolar Injuries
- ▶ Mandibular Fractures
- ▶ Maxillary Fractures
- ▶ Nasal and Zygomaticomaxillary Fractures
- ▶ Naso-Orbito-Ethmoid (NOE) and Frontal Sinus Fractures
- ▶ Orbital Fractures
- ▶ Special Considerations
- ▶ Firearm Injuries to Head and Neck
- ▶ Management of Complications

Clinical Evaluation

All patients should undergo a proper advanced trauma life support (ATLS) assessment on arrival at the emergency room. This is usually performed by the emergency trauma team. The basic initial assessment should follow the ATLS sequence:

1. Primary survey
2. Shock evaluation
3. Emergency airway management
4. Chest trauma evaluation
5. Neurologic evaluation, including the level of consciousness and calculation of a Glasgow Coma Scale; a value of less than 8 is usually considered a marker for intubation
6. Abdominal evaluation

Glasgow Coma Scale

Eye opening		Motor response		Speech	
Spontaneous	4	Follows commands	6	Oriented	5
To speech	3	Localizes pain	5	Disoriented	4
To pain	2	Withdraws to pain	4	Inappropriate	3
No response	1	Abnormal flexion	3	Incomprehensible	2
		Abnormal extension	2	No response	1
		No response	1		

An oral and maxillofacial surgeon's primary responsibilities during the initial stages of trauma assessment are:

- Clearing the airway of debris, loose teeth, removable dental appliances, and unsalvageable bony fragments that may pose a threat to maintenance or restoration of the airway
- Temporary stabilization of a repositioned mandible secondary to bilateral condylar and/or symphysis fracture(s)
- Control of hemorrhage with nasal packing, pressure dressings on facial lacerations, and/or clamping of accessible hemorrhaging vessels

Cranial Nerve (CN) Quick Assessment Guide

CN	Function	Physical finding
I	Sense of smell	Anosmia, hyposmia, or cacosmia
II	Changes in light perception and/or visual acuity; evaluate using handheld flashlight and visual acuity chart	Ophthalmoplegia: blindness (partial or complete), visual field deficits, blurring, or scotomata
III	Pupillary reaction to light, ocular motility	<ul style="list-style-type: none"> • Outward and downward deviation of the eye • Ptosis of the eyelid • Dilation of the ipsilateral pupil in complete palsy
IV	Ocular motility in medial gaze	Vertical diplopia on looking downward, which improves with contralateral head tilt and worsens with ipsilateral head tilt
V	All three divisions of the nerve are responsible for touch, temperature, and motor function	Paresthesia; evaluate and record any deficits
VI	Ocular motility in lateral gaze	<ul style="list-style-type: none"> • In a complete injury of the abducens nerve, the affected eye is turned medially • In an incomplete injury, the affected eye is seen in the midline at rest, but the patient cannot move the eye laterally
VII	Motor nerve to muscles of facial expression	Complete or partial paralysis of the face, hyperacusis, and/or an unusual or impaired sense of taste can occur after injury
VIII	Hearing and balance	<ul style="list-style-type: none"> • Positional vertigo is the most common problem, although tinnitus, hearing loss, and deafness may also occur • Check for otorrhea and hearing loss
IX	Taste from posterior third of tongue, parotid secretion	Difficult to examine in acutely injured patients
X	Branchiomotor innervation to most laryngeal and pharyngeal muscles	Vocal cord changes, dysphagia
XI	Innervates SCM and trapezius muscles	Difficulty to move and rotate head
XII	Tongue motor innervation	Tongue protrudes to ipsilateral side
SCM, sternocleidomastoid.		

Classification of Hemorrhage

	I	II	III	IV
Blood loss (mL)	< 750	750–1,500	1,500–2,000	> 2,000
Blood loss (%)	< 15	15–30	30–40	> 40
Pulse rate (beats/min)	< 100	100–120	120–140	>140
Blood pressure	Normal	Decreased	Decreased	Decreased
Respiratory rate (breaths/min)	14–20	20–30	30–40	> 35
Urine output (mL/hr)	> 30	20–30	5–15	Negligible
Central nervous system	Normal	Anxious	Confused	Lethargic

Physical Examination

Scalp

Palpate the hair-bearing scalp, where injuries may be concealed, and examine it for evidence of bleeding. Hematomas that require evacuation may also be present.

Ocular structures

- Direct and indirect pupillary reflexes need to be evaluated using a handheld light source
- Visual acuity analyzed using a Snellen chart
- Confrontation visual field examination
- Slit-lamp ocular examination
- Intraocular tonometry (normal 10–21 mm Hg)
- Schirmer tear test
- Hertel measurement, enophthalmos; Hertel exophthalmometer is the instrument most frequently used to measure the position of the globe in relation to the orbit.

Signs and symptoms of ocular trauma

	Definition	Possible causes	Management
Abnormal pupillary reflexes	Normal pupillary size using a conventional penlight should be between 2.6 and 3.6 mm depending on the light conditions of the room	Optic nerve damage, oculomotor nerve damage, brain stem injuries, drugs, or death	Depends on cause; observation or further imaging
Anisocoria	Unequal pupil size	Physiologic, Horner syndrome, mechanical, nerve palsy, drugs	Depends on cause; observation or further imaging
Marcus Gunn syndrome	Unilateral nonreactive pupil that exhibits contralateral pupillary constriction when light is shined into it	Optic nerve damage or retinal disease	Further imaging and/or work-up
Muscle entrapment	Inability to move the eye in a particular gaze	Fracture, edema, nerve injury	Observation or surgical release
Ecchymosis	Bruising	Bleeding	Local heat and observation
Hyphema	Blood in the anterior chamber	Fracture, blunt trauma	Observation
Chemosis	Swelling or edema of the conjunctiva	Infection, trauma, allergic reaction	Observation
Diplopia	Double vision	Fracture, edema, nerve injury	Depends on etiology
Proptosis	Bulging of the eye anterior to the orbit	Hematoma, edema, fracture, systemic disease	Depends on etiology
Retrobulbar hematoma	Intraorbital bleeding	Progressively tense and painful proptosis, decreased ocular motility, asymmetric visual acuity, field restriction on confrontation testing, decreased pupil responsiveness	Lateral canthotomy
Superior orbital fissure syndrome	Dysfunction of CN III, IV, V (v1/v2), VI	Fractured bony segment or hematoma	Depends on etiology
Orbital apex syndrome	Same as superior orbital fissure syndrome but with optic nerve injury	Fractured bony segment or hematoma	Depends on etiology
Traumatic telecanthus	Increased intercanthal distance (> 35 mm)	Disruption of attachment of medial canthal tendon	ORIF of attached segment of bone or transnasal wiring

ORIF, open reduction and internal fixation.

Nasal cavity

- Nasal bone stability. Bimanually examine the bony and cartilaginous framework.
- Nasal speculum examination. Inspect the intranasal structures for septal deviation or hematomas.

Signs and symptoms of nasal fracture

	Definition	Possible cause	Management
Epistaxis	<ul style="list-style-type: none"> • From the anterior vascular plexus (Kiesselbach) • From the posterior vascular plexus (Woodruff) 	Trauma, bleeding disorder	<ul style="list-style-type: none"> • Nasal pack (foam and/or strips) • Foley catheter • Electrocautery • Coagulation sticks
Cerebrospinal fluid (CSF) leak	Clear rhinorrhea <ul style="list-style-type: none"> • Ring sign on filter paper • Beta-2 transferrin test • Lab testing <ul style="list-style-type: none"> – Higher chloride (113 vs 99 mEq/L) – Lower glucose (60 vs 100 mEq/L) 	Anterior cranial base fracture and dural tears	Consult neurosurgeon
Septal hematoma	Blood collection between perichondrium of nasal septum and the septal cartilage	Trauma	Considered an emergency: Aspirate with large-bore needle or drain and then pack nose immediately

Ear

- Complete ear inspection for cartilaginous injuries, lacerations, hematomas, or otorrhea
- Otoscopic examination of the middle ear structure
- Tuning fork to determine CN VIII compromise and vertigo

Ear-related signs and symptoms of basilar skull fracture

	Definition	Possible cause	Management
Battle sign	Bruising along mastoid process	Basilar skull fracture	Elevate bed 45 degrees, sinus precautions
Otorrhea	Drainage of fluid from ear (CSF)	Basilar skull fracture	Elevate bed 45 degrees, sinus precautions
Hemotympanum	Blood in the tympanic cavity of the middle ear	Basilar skull fracture	Elevate bed 45 degrees, sinus precautions

Maxillofacial complex

- Inspect the oral cavity for debris, loose tooth segments, or lacerations
- Evaluate the tongue for lacerations, bleeding, or edema that may obstruct the airway
- Evaluate residual dentition and determine the feasibility of placing arch bars, the need for fabrication of splints, and the type of intubation desired
- Account for any missing teeth and correlate later with radiographic evaluation

Signs and symptoms of injury to the maxillofacial complex

	Possible causes	Management
Malocclusion	<ul style="list-style-type: none"> • Dentoalveolar fracture • Maxillary/mandibular fracture 	Bridle wire, soft diet
Hematoma in floor of mouth	<ul style="list-style-type: none"> • Mandibular fracture • Injury to the lingual vessels 	Hospital admission for airway observation
Maxillary vestibular ecchymosis	Maxillary fracture	Soft diet, ORIF
ORIF, open reduction and internal fixation.		

Neck

- The trachea should be in midline
- Blunt trauma to the neck or gunshot wound may require evaluation with a computed tomography (CT) angiogram

Blunt neck trauma

	Location	Management
Zone 1	Inferior aspect of cricoid cartilage to the thoracic outlet	CT angiogram and exploration
Zone 2	Cricoid to angle of mandible	CT angiogram and exploration
Zone 3	Angle of mandible to the base of the skull	CT angiogram and exploration

Radiographic Examination

	Indications
Dental radiograph	Dentoalveolar trauma
Panoramic radiograph	Mandibular/dentoalveolar trauma; two-dimensional view helpful in assessment of dentition and mandibular trauma
CT scan, noncontrast	Polytrauma patient; scan of the maxillofacial region with coronal, sagittal, and axial cuts of no more than 2 mm in thickness
CT angiogram	<ul style="list-style-type: none"> Firearm injuries of the head and neck Penetrating trauma or any actively bleeding neck wound that does not stop with local measures
CBCT reconstruction	Polytrauma patient; helpful in midface, naso-orbito-ethmoid (NOE), and frontal bone trauma
CBCT, cone beam computed tomography.	

Anesthesia in the Traumatized Patient

Intubation

	Indication	Description
Nasal	Mandibular and/or maxillary fractures	Preferred method when treating facial fractures due to the necessity of using maxillomandibular fixation (MMF) during the operation
Oral	Zygomaticomaxillary complex (ZMC), orbital, or frontal bone fractures	When there are no changes in occlusion, oral intubation is preferred
Submental	Mandibular and/or maxillary fractures when nasal intubation is not possible	<ul style="list-style-type: none"> A metal-reinforced endotracheal tube is recommended to prevent collapse Caution should be used to stay within proximity of mandibular lingual cortex to avoid injury to a lingual vein or submandibular duct

Tracheotomy

Indications	<ul style="list-style-type: none"> Nasal or oral intubation is not possible Patient is expected to be intubated for more than 7 days
Skin incision design	<ul style="list-style-type: none"> Horizontal: More cosmetic, but is not recommended in emergency situations because of increased risk of vascular injury Vertical: Less cosmetic, and recommended in emergency situations
Pertinent anatomy	<ul style="list-style-type: none"> Layers of skin, subcutaneous tissue, strap muscles of the neck, and the thyroid isthmus At the midline there is very little risk of injury to vessels or nerves during the dissection The thyroid isthmus can be tied off or cauterized The anterior jugular veins and/or arteries can be tied or retracted
Tracheal incision design	<ul style="list-style-type: none"> Vertical incision placed between the second and third tracheal rings Björk incision, an inferiorly based flap through second and third tracheal rings
Tracheostomy tube	<ul style="list-style-type: none"> Cuffed, noncuffed, fenestrated 6-0 or 8-0

Common Extubation Criteria

Ventilation	<ul style="list-style-type: none"> Negative inspiratory force (NIF) more than -30 cm H_2O Forced ventilatory capacity (FVC) greater than or equal to 10 L/sec Peak expiratory flow rate (PEFR), the higher the better Minute volume less than 10 Lpm in adults Respiratory rate less than 35 breaths per minute in adults
Laboratory results	<ul style="list-style-type: none"> Stable partial pressure of oxygen (PaO_2): 80 to 100 mm Hg Stable partial pressure of carbon dioxide ($PaCO_2$): 34 to 45 mm Hg pH between 7.35 and 7.45
Clinical findings	<ul style="list-style-type: none"> Minimal secretions Alert, cooperative Minimal work for breathing Stable cardiovascular status

Principles of Fixation

Plate Materials

Titanium alloy	<ul style="list-style-type: none">• Most commonly used material• More strength than grade 4 pure titanium
Stainless steel	<ul style="list-style-type: none">• Contains nickel• Caution in patient with nickel allergy• Commonly used for MMF screws
Resorbable material	<ul style="list-style-type: none">• Converted by human body into CO₂ and H₂O• More bulky than titanium plates

Screw Anatomy

The size determines the system. A 2.0 system is designed for a plate that fits 2.0-mm screws. It does not indicate the thickness of the plate. Screws are classified based on size, drilling mechanism, and head configuration.

Size	Determined by the outer diameter and measured in millimeters <ul style="list-style-type: none">• Midface: 1.2–1.7 mm• Mandible: 2.0–2.7 mm
Thread shape	<ul style="list-style-type: none">• Self-tapping: Two or three cutting flutes for insertion with predrilling but no tapping• Self-drilling: One or two cutting flutes for insertion without predrilling or tapping
Head configuration	<ul style="list-style-type: none">• Locking screw: Screw tightens into plate creating one rigid unit; does not require the plate contour to fit the bone perfectly• Nonlocking screw: If the plate is not well contoured to the bone, it displaces bone segments; micromovement of the bone leads to screw loosening
Lag screw	<p>Advantages</p> <ul style="list-style-type: none">• Provides absolute rigid fixation• Requires no additional hardware• Provides compression• Great for symphysis fractures <p>Disadvantages</p> <ul style="list-style-type: none">• Requires strong and/or stable bony segments• Improper reduction leads to malocclusion and shortened and/or overriding fracture alignment

Plate Design

- Plate profile determines the thickness of the plate
- Static design has round, recessed holes that accept neutral screws
- Dynamic compression design has spherical screw holes that permit screw placement at an angle; when screws are inserted eccentrically, the plate compresses the fracture

Bone Plates and Screws for Maxillofacial Surgery

Metallic fixation devices of various thicknesses and materials are essential for treating maxillofacial fractures.

	Orbital trauma	Nasal fractures	Maxillary pillars	Mandible fractures	Reconstruction
Thickness (mm)	0.5 to 0.7	0.5 to 1.0	0.6 to 1.0	1.0 to 3.0	2.0 to 3.0
Screw size (mm)	1.3 to 1.5	1.3 to 2.0	1.5 to 2.0	1.6 to 2.7	2.0 to 2.7

Biomechanical Concepts

Load-sharing versus load-bearing

Load-sharing

- Adequate bone stock at fracture site
- Use miniplate to achieve stability
- Force is shared between the hardware and the bony segments

Load-bearing

- Inadequate bone stock (ie, atrophic mandible, continuity defect, or comminution) at fracture site
- Use reconstruction plate (2.0- to 3.0-mm thickness) with a minimum of three screws on each side of the defect/fracture

Functionally stable versus rigid fixation

Functionally stable

- Firm fixation but micromovement is allowed during function
- Functional forces are shared between the hardware and the bone
- Less hardware is needed
- Fixation is nonrigid but stable enough to obviate the need for postoperative MMF

Rigid fixation

- Fixation with no micromovement during function
- Strong but may require more hardware
- Follow Academy of Osseointegration principles
 - Anatomical reduction
 - Stable fixation
 - Preservation of the blood supply
 - Early mobilization

MMF screws versus arch bars

MMF screws

- Advantages: Ease of placement, nondental-dependent skeletal fixation
- Disadvantages: May traumatize dental roots, increased cost, not acceptable for comminuted fractures

Arch bars

- Advantages: Economical, acceptable for comminuted fractures, can serve as a superior border tension band
- Disadvantages: Depends on status of existing dentition, longer operating room time, anesthesia may be needed for arch bar removal, may cause extrusion of teeth

Soft Tissue Injuries

Biologic Healing Properties of the Face

Characteristics of biologic healing in the maxillofacial region

- Different from the extremities because of richer vasculature, which leads to more rapid healing and fewer infections
- Minimal debridement is usually required
- Many tissue fragments that look avascular will survive after reapproximation and stabilization

Stages of wound healing	
Inflammation	Heat, erythema, edema, and recruitment of mast cells, neutrophils, and macrophages
Proliferation	Formation of granulation tissue, mainly endothelial cells and fibroblasts
Remodeling	Increase in wound strength and collagen remodeling

Sequence of Management

- Local anesthesia alone can be used in most instances of soft tissue injuries in the face; regional blocks of the trigeminal nerve branches will allow the clinician to work on the awake patient
- The most common anesthetic used is lidocaine with the addition of epinephrine to act as a vasoconstrictor and help with hemostasis
- For longer procedures, bupivacaine is recommended because of its longer duration of action

Primary management	<p>Guidelines</p> <ul style="list-style-type: none"> • Primary closure within the first 24 hours of injury • Delayed primary closure (24 to 72 hours) is indicated when the wound is extremely contaminated and the risk of infection is high • The wound is debrided, irrigated, and cleansed several times and closed after parenteral antibiotics have been started <p>Irrigation and debridement</p> <ul style="list-style-type: none"> • Irrigation requires pressure of 7 psi in order to remove adherent bacteria from a wound • At least 1 L of either sterile saline or a 2:1 solution of saline and povidone-iodine should be used • A scrub brush is helpful, especially for abrasions <p>Wound closure</p> <ul style="list-style-type: none"> • Use 3-0 to 5-0 resorbable suture (eg, Monocryl or Vicryl [Ethicon]) to reapproximate deep tissues • Skin closure may be done with 5-0 or 6-0 absorbable suture (ie, fast-absorbing gut) for pediatric patients or permanent suture (ie, nylon or Prolene [Ethicon]) for other patients • If skin edges are precisely approximated, 2-octyl-cyanoacrylate may be applied for small, shallow lacerations • Avoid performing local flaps in the “primary” setting
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(Sequence of Management cont)

Secondary management	<ul style="list-style-type: none">• Scar revision should be performed after full maturation of the wound has occurred (usually between 6 and 12 months)• Intralesional steroid-like triamcinolone is recommended (10 mg/mL) to help with hypertrophic scar formation and to soften the edges of the wound
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Special Considerations

Animal bites	<ul style="list-style-type: none">• Cause infection in approximately 20% of cases; more infection associated with human bites > cat bites > dog bites• Most common bacteria are <i>Pasteurella</i> or <i>Capnocytophaga</i> species• Adequate cleaning and irrigation, in conjunction with antibiotics, should be the initial treatment of choice• Amoxicillin and clavulanate (Augmentin [GlaxoSmithKline]) is the recommended anti-biotic, but only for cat and human bites																		
Tetanus immunization	<ul style="list-style-type: none">• Two types of tetanus injection<ul style="list-style-type: none">– Active immunization with tetanus toxoid (TT)– Passive immunization with human tetanus immune globulin (HTIG)• Wounds more prone to tetanus<ul style="list-style-type: none">– Treatment delayed for more than 6 hours– Deep puncture– Avulsion– Heavy contamination <table><tr><th colspan="3">Patient history of tetanus immunization</th></tr><tr><th>Time since last immunization</th><th>TT</th><th>HTIG</th></tr><tr><td>None or unknown</td><td>Yes</td><td>Consider</td></tr><tr><td>< 5 years</td><td>No</td><td>No</td></tr><tr><td>5–10 years</td><td>Booster</td><td>No</td></tr><tr><td>> 10 years</td><td>Booster</td><td>Consider</td></tr></table>	Patient history of tetanus immunization			Time since last immunization	TT	HTIG	None or unknown	Yes	Consider	< 5 years	No	No	5–10 years	Booster	No	> 10 years	Booster	Consider
Patient history of tetanus immunization																			
Time since last immunization	TT	HTIG																	
None or unknown	Yes	Consider																	
< 5 years	No	No																	
5–10 years	Booster	No																	
> 10 years	Booster	Consider																	
Traumatic facial nerve injury	<ul style="list-style-type: none">• Second most common cause of facial nerve paralysis after Bell palsy• Causes<ul style="list-style-type: none">– Temporal bone fracture (most common traumatic cause)– Penetrating trauma• Treatment<ul style="list-style-type: none">– Distal to the zone of arborization (line perpendicular to the lateral canthus), observation and steroids– Proximal to the zone of arborization, surgical exploration, approximation if possible, and steroid																		

(Special Considerations cont)

Parotid duct injury	<ul style="list-style-type: none">• Stensen duct<ul style="list-style-type: none">– Runs parallel with the buccal branch of facial nerve (inferior to the duct)– Follows a line drawn from the tragus to the midportion of the upper lip• Treatment if both ends are present<ul style="list-style-type: none">– Anastomosis– A silastic stent should be placed and sutured to the mucosa for 2 to 4 weeks to prevent accidental displacement• Treatment if only one end is present or both ends are missing<ul style="list-style-type: none">– A pressure dressing is used to prevent sialocele– Multiple aspirations of accumulated saliva may be needed– Oral glycopyrrolate may be needed– Persistent swelling may require low-dose radiation therapy
Lacrimal duct injury	<ul style="list-style-type: none">• Occurs in 5% of NOE fractures• Prophylactic management is not indicated• If a tear between the punctum and the lacrimal sac is noticed<ul style="list-style-type: none">– Intubation with a Crawford tube for 12 to 16 weeks (shorter time in pediatric patients)– Dacryocystorhinostomy is indicated if chronic epiphora develops
Lip injury	Primary closure is possible for a defect that affects up to $\frac{1}{4}$ of the upper lip or $\frac{1}{3}$ of the lower lip; a local or regional flap is necessary if a lip defect is any larger (see chapter 8)
Ear injury	Cartilage is present only in the upper $\frac{2}{3}$ of the ear; a superficial defect can be managed with <ul style="list-style-type: none">• Healing by secondary intention: The presence of underlying cartilage prevents distortion from wound contraction• Full-thickness skin graft, but only if the perichondrium is present (For discussion of a large aural defect, see chapter 8.)

Dentoalveolar Injuries

	Description	Management
Concussion	Injury to the tooth-supporting structures without mobility of the tooth but with pain to percussion	<ul style="list-style-type: none">• Observe• Monitor pulpal condition and root resorption for at least 1 year with clinical and radiographic examinations
Subluxation	Injury to the tooth-supporting structures resulting in increased mobility but without displacement of the tooth	<ul style="list-style-type: none">• Place flexible splint for 2 weeks• Monitor pulpal condition and root resorption for at least 1 year with clinical and radiographic examinations

(Dentoalveolar Injuries cont)

	Description	Management
Extrusion	Injury to the tooth characterized by partial or total separation of the periodontal ligament resulting in loosening, displacement, and an element of protrusion	<ul style="list-style-type: none"> • Clean the root surface with saline before repositioning with axial digital pressure • Stabilize the tooth for 2 weeks using a flexible splint • Monitor pulpal condition and root resorption for at least 1 year with clinical and radiographic examinations
Lateral luxation	Displacement of the tooth other than axially	<ul style="list-style-type: none"> • Perform digital repositioning • Stabilize for 4 weeks • Monitor pulpal condition and root resorption for at least 1 year with clinical and radiographic examinations
Intrusion	Displacement of the tooth into the alveolar bone; potential risk of tooth loss due to progressive root resorption (ankylosis or infection-related resorption)	<ul style="list-style-type: none"> • Endodontic treatment can prevent the necrotic pulp from initiating infection-related root resorption • Endodontic therapy should preferably be initiated within 3 to 4 weeks posttrauma • A temporary root canal filling with calcium hydroxide is recommended
Avulsion	Complete displacement of the tooth out of the socket	<ul style="list-style-type: none"> • Clinical management is dictated by “dry time” and apex condition • Treatment within 60 minutes or less: Place a flexible splint for 2 weeks • Treatment after more than 60 minutes: Poor prognosis or ankylosis • With open apex cases, initial endodontics is not recommended • With closed apex cases, endodontic treatment is always recommended
Alveolar fracture	A fracture of the alveolar process; may or may not involve the tooth socket	<ul style="list-style-type: none"> • Reposition segment • Stabilize for 4 weeks

Mandibular Fractures

These are the most common fractures in the face after nasal bone fractures. The most common mandibular fracture is a combination of an angle fracture with a contralateral body or symphysis fracture. Most mandible fractures occur in pairs.

- Biomechanics
 - Compression zone
 - Tension zone
 - Neutral zone

- Mandibular fractures are usually classified by
 - Location
 - Communication with the oral cavity or skin: Open or closed
 - Complete or incomplete
 - Displacement: Minimal, moderate, severe
 - Type: Linear versus comminuted
 - Mobility: Mobile or nonmobile
 - Duration: Acute (< 3 weeks) or chronic (> 4 weeks)

Fracture Variables

The following variables must be identified when deciding the type of repair required.

Dentition	<ul style="list-style-type: none">• Dentate patient: Arch bar can be used as a superior border tension band• Edentulous patient<ul style="list-style-type: none">– > 15 mm of mandibular height is nonatrophic– < 15 mm of mandibular height is atrophic<ul style="list-style-type: none">◦ Requires reconstruction plate◦ Bilateral fractures are common• Absence of teeth distal to the fracture<ul style="list-style-type: none">– MMF will not stabilize the segments, and the elevator muscles tend to rotate the proximal segment upward– Open reduction and internal fixation (ORIF) is recommended
Teeth in the line of fracture	<ul style="list-style-type: none">• Indications for extraction<ul style="list-style-type: none">– Advanced caries and/or periodontal disease– Pathology– Midroot fracture– Preventing proper reduction• Extraction may decrease the available bone for subsequent implant placement
Number of fractures	<ul style="list-style-type: none">• Usually the higher the number of fractures, the greater the likelihood of needing open reduction• Contrary to popular belief, comminution should not be a contraindication for ORIF
Infection	<ul style="list-style-type: none">• Acute: Treated similarly to noninfected sites• Chronic<ul style="list-style-type: none">– Requires load-bearing fixation– Screws should not be placed in contaminated or osteoporotic areas– Simultaneous bone grafting can be considered especially in the osteoporotic mandible

Principles of Repair

All fractures treated by open reduction should be exposed and reduced temporarily with the help of clamps or circumdental wiring prior to establishing the occlusion, especially in the case of multiple fractures.

Anatomical unit	Description
Temporomandibular joint contusion	<ul style="list-style-type: none"> • Closed reduction • Observation and soft diet • Subsequent aggressive physical therapy is paramount to return the patient's normal range of motion
Condylar fracture	<ul style="list-style-type: none"> • Indications for open reduction and internal fixation <ul style="list-style-type: none"> – Bilateral displaced condylar fractures – Combined condylar fractures with associated mobile midface fractures – Edentulous mandibles – Any other situation where the clinician feels the outcome with open treatment would be better than with closed treatment • Strong fixation is needed to prevent rotational movement <ul style="list-style-type: none"> – Two 2.0-mm miniplates – One rigid 2.0-mm plate
Body fracture	<ul style="list-style-type: none"> • Usually combined with fractures on the contralateral side or with ipsilateral fractures in the ramus or condyle region • Treatment options <ul style="list-style-type: none"> – Closed reduction if there are teeth on both sides of the fracture – Miniplates at the tension and compression sites – Reconstruction plate along the inferior border – Arch bar as a tension band and one miniplate in the compression zone
Angle fracture	<ul style="list-style-type: none"> • MMF does not usually work for angle fractures; the proximal segment tends to rotate upward from the pull of the elevator muscles • Treatment options <ul style="list-style-type: none"> – Single miniplate with the Champy technique; preferred due to fewer major complications – Plates in the tension and compression sites – Reconstruction plate on the inferior border
Symphysis fracture	<ul style="list-style-type: none"> • Isolated fractures can be treated with closed reduction if there are teeth on both sides of the fracture • Treatment options if ORIF is chosen <ul style="list-style-type: none"> – Two miniplates – Two lag screws – Reconstruction plate along the inferior border – Arch bar and one miniplate

(Principles of Repair cont)

Atrophic mandible	<ul style="list-style-type: none">• < 15 mm height• Load-bearing reconstruction plate via extraoral approach• Immediate bone grafting should be considered to promote adequate bony union, especially in elderly patients
Multiple fractures	<ul style="list-style-type: none">• For fractures located in close proximity, use MMF, or placement of a reconstruction plate along the inferior border will be adequate• For fractures located far apart<ul style="list-style-type: none">– Treat anterior before posterior (non-tooth bearing)– Multiple miniplates or one reconstruction plate

Complications

Nonunion	<ul style="list-style-type: none">• Can occur after any ORIF of fractures• More common in mandible due to mobility of the jaw• Usually the consequence of infection, hardware failure, or micromotion• Treatment<ul style="list-style-type: none">– Remove infected or contaminated hardware– Debride granulation tissue– Closed reduction or ORIF with new hardware– An autogenous bone graft is recommended in most cases
Malunion	<ul style="list-style-type: none">• Secondary to improper MMF before final fixation• May be secondary to condylar fracture that could not be successfully treated• Treatment<ul style="list-style-type: none">– Surgically reopen the fracture or perform standard osteotomies– Reestablish the proper occlusion– Stabilize the bone segments with internal fixation device
Osteomyelitis	<ul style="list-style-type: none">• Requires the use of systemic antibiotics for 6 weeks via peripherally inserted central catheter• Removal of hardware• Subsequent debridement and bone grafting• Improper management can lead to<ul style="list-style-type: none">– Orocutaneous fistula– Meningitis– Cavernous sinus thrombosis
Cervical spine fracture	One in 50 mandibular fractures associated with subluxation of the cervical spine

Maxillary Fractures

Vertical Buttresses

- Ramus-condyle
- Lateral zygomaticomaxillary
- Medial nasomaxillary

Horizontal Buttresses

- Supraorbital
- Infraorbital
- Maxillary alveolar
- Mandibular body

Le Fort Fractures

- Can be of different types bilaterally

Le Fort I (Fig 6-1)

Treatment

- If no malocclusion, conservative treatment (ie, soft diet, antibiotics, follow-up)
- Malocclusion
 - If occlusion can be established by digital manipulation: MMF
 - If occlusion cannot be established by digital manipulation: ORIF

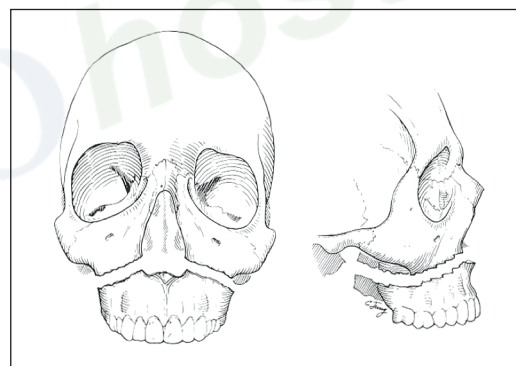


Fig 6-1 Le Fort I fracture.

Le Fort II (Fig 6-2)

Treatment

- If no malocclusion, conservative treatment (ie, soft diet, antibiotics, follow-up)
- Malocclusion
 - If occlusion can be established by digital manipulation: MMF
 - If occlusion cannot be established by digital manipulation: ORIF

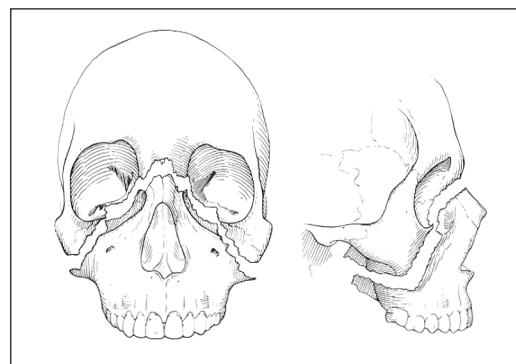


Fig 6-2 Le Fort II fracture.

Le Fort III (Fig 6-3)

Treatment

- Coronal, intraoral, and possibly lower lid approaches are required
- Plates must be placed across the frontozygomatic sutures and frontonasal areas to help stabilize the midface
- Internal orbital reconstruction will often be necessary
- Simultaneous bone grating should be considered in areas of large defects to promote adequate and stable healing

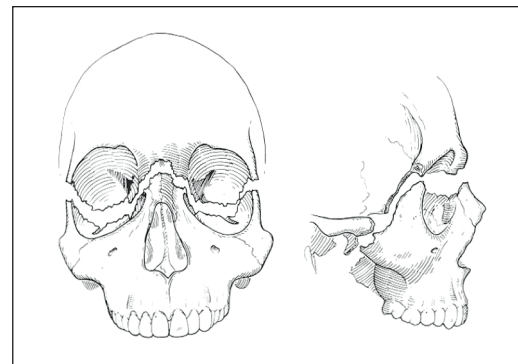


Fig 6-3 Le Fort III fracture.

Nasal and Zygomaticomaxillary Fractures

Nasal Bone Fractures

Incidence	<ul style="list-style-type: none"> • Most common facial fracture
Diagnosis	<ul style="list-style-type: none"> • Physical examination and CT scan without contrast
Physical findings	<ul style="list-style-type: none"> • Pain, crepitation, septal hematoma, nasal deviation, epistaxis (from Kiesselbach and/or Woodruff plexuses)
Complications	<ul style="list-style-type: none"> • Septal hematoma: Must be immediately drained and nose packed; untreated can lead to septal perforation • Epistaxis: Cautery and/or packing • Septal deviation • Saddle nose deformity
Treatment	<ul style="list-style-type: none"> • No deformity: Pain medication and antibiotics • Deformity present <ul style="list-style-type: none"> – Closed reduction under local anesthesia and/or intravenous sedation or general anesthesia – Open reduction under general anesthesia within 1 week or after swelling subsides

Zygomaticomaxillary Complex (ZMC) Fractures

Anatomy	Involves four areas (tetrapod) <ol style="list-style-type: none"> 1. Zygomaticofrontal (ZF) suture 2. Zygomaticomaxillary (ZM) buttress <ul style="list-style-type: none"> – Greatest surface area, most reliable in determining the accuracy of reduction 3. Zygomaticotemporal (ZT) suture 4. Zygomaticosphenoid (ZS) suture
Diagnosis	<ul style="list-style-type: none"> • Physical examination • Plain radiograph (Waters view) • CT scan without contrast
Clinical findings	<ul style="list-style-type: none"> • Functional <ul style="list-style-type: none"> – Ocular muscle entrapment – Diplopia – Enophthalmos – Hypophthalmos – Trismus – Malocclusion – Infraorbital paresthesia • Cosmetic <ul style="list-style-type: none"> – Globe malposition – Malar retrusion – Facial asymmetry
Classification	Zingg classification based on CT scans and mechanism of injury <ul style="list-style-type: none"> • Type A: Isolated to one component of tetrapod <ul style="list-style-type: none"> – A1: Zygomatic arch – A2: Lateral orbital wall – A3: Inferior orbital rim • Type B: All four areas of tetrapod • Type C: Complex with comminution of zygomatic bone
Treatment	Closed reduction <ul style="list-style-type: none"> • Used if no orbital floor reconstruction is necessary and manual manipulation into normal position is possible • Two common methods <ul style="list-style-type: none"> – Gillies approach – Keen approach (intraoral incision) Open reduction <ul style="list-style-type: none"> • Expose ZM buttress intraorally and place plate • If still unsure of reduction, expose ZF and ZT sutures through skin incisions and place plate • Fix at least two areas to ensure proper reduction • If orbital reconstruction is needed, a lower lid incision is used after ZMC reduction and plating

Naso-Orbito-Ethmoid (NOE) and Frontal Sinus Fractures

NOE Fractures

Anatomy	Nasolacrimal duct drains into inferior meatus (covered by valve of Hasner)
Clinical findings	<ul style="list-style-type: none"> • Telecanthus: Medial intercanthal distance $> \frac{1}{2}$ interpupillary distance (normal 30 to 35 mm) • Rounding of the medial canthus • Shortened palpebral fissure • Saddle nose deformity (loss of nasal projection or height and flattened dorsum) • Rhinorrhea • Epiphora • Infraorbital paresthesia • Positive bowstring test: The skin of the eyelid is pulled laterally to detect mobility of the frontal process of the maxilla
Classification	Markowitz classification <ul style="list-style-type: none"> • Type I: Single large NOE fragment bearing the medial canthal tendon • Type II: Comminution of the NOE area, but the canthal ligament remains attached to a fragment of bone • Type III: Comminution of the NOE area and detachment of the medial canthal ligament from the bone
Treatment	<ul style="list-style-type: none"> • The key to the treatment of NOE fractures is proper reduction of the medial canthal ligament; attach to medial orbital wall or frontal process of the maxilla; the fragments are usually laterally displaced – Type I: Usually can be reduced without difficulty <ul style="list-style-type: none"> – Type II: Canthal ligament can be stabilized on the fractured segment in a posterior and superior direction with wires or a small plate – Type III: Requires canthopexy • The bony nasal dorsum will generally also require reconstruction with a cortical bone graft, usually harvested from the skull

Frontal Sinus Fractures

Incidence	Occurs in 5% to 15% of all facial fractures
Indications for treatment	<ul style="list-style-type: none"> • Functional <ul style="list-style-type: none"> – Blockage of nasofrontal duct – CSF leaks from posterior table fractures; at risk of meningitis • Cosmetic: Frontal depression from anterior table fracture
Anatomy	<ul style="list-style-type: none"> • Frontal sinus missing at birth in 5% of population • Radiographically identifiable by 6 to 8 years of age • Fully pneumatized by 12 to 16 years of age • For 85% of the population, the nasofrontal sinus drains directly into middle meatus (hiatus semilunaris) via an ostium • Breschet canals (foramina): Venous structures in the posterior table of the frontal sinus with direct access into anterior cranial fossa • Only 15% of the population has a true nasofrontal duct
Diagnosis	<ul style="list-style-type: none"> • Physical examination and CT without contrast
Clinical findings	<ul style="list-style-type: none"> • Periorbital ecchymosis • CN V1 paresthesia • Forehead deformity • Rhinorrhea • Pneumocephalus on CT scan indicates posterior wall involvement
Treatment	<ul style="list-style-type: none"> • Based on <ul style="list-style-type: none"> – Type: Anterior wall, posterior wall, combination – Amount of displacement – Status of nasofrontal duct • Evaluation of nasofrontal duct patency <ul style="list-style-type: none"> – Preoperative: Coronal view CT scan – Intraoperative: Injection of dye (methylene-blue or fluorescein) into the nasofrontal duct • Frontal sinus obliteration <ul style="list-style-type: none"> – Indications <ul style="list-style-type: none"> ◦ Posterior table involvement ◦ Nasofrontal duct obstruction – Mucosa is removed by manual curettage and rotary instruments; should include the sinus mucosa that is invaginated into the vascular pits of Breschet – Material for obliteration <ul style="list-style-type: none"> ◦ No one material is superior to another ◦ Commonly used materials: Abdominal fat, temporalis muscle, pericranium

(Frontal Sinus Fractures cont)

Complications	<ul style="list-style-type: none"> • Early <ul style="list-style-type: none"> – Meningitis – Limited eye movement: Trauma to the superior oblique muscle or trochlear nerve • Chronic: Mucocele, which can take 20 years to develop
Follow-up	<ul style="list-style-type: none"> • Periodic CT sinus scans (1, 2, and 5 years) and whenever symptoms appear

Fracture type	Displacement	Nasofrontal duct	Management
Anterior table	None	NA	Observation and sinus precautions
	Displaced	Not obstructed	ORIF and sinus precautions
	Displaced	Obstructed	ORIF and obliteration
Posterior table (always together with anterior table)	None	NA	Observation and sinus precautions
	Displaced	Always obstructed	ORIF, dural repair and cranialization, and nasofrontal duct obliteration

Orbital Fractures

Anatomy

Upper eyelid layers	Lower eyelid layers
<ul style="list-style-type: none"> • Skin • Subcutaneous tissue • Orbicularis oculi • Orbital septum • Tarsal plate (10-mm height) and levator aponeurosis above tarsal plate • Muller muscle • Conjunctiva 	<ul style="list-style-type: none"> • Skin • Subcutaneous tissue • Orbicularis oculi • Orbital septum • Tarsal plate (5-mm height) • Conjunctiva

Ligaments

Whitnall ligament (superior transverse ligament)	<ul style="list-style-type: none"> • 14 to 20 mm from the upper tarsal plate • Function: Suspends the levator aponeurosis and the medial portion of the superior oblique tendon • Injury: Ptosis of the nasal portion of the upper lid • Composed of <ul style="list-style-type: none"> – Levator muscles – Superior oblique tendon • Attaches to orbital lobe of the lacrimal gland • Levator muscles pass through Whitnall ligament and become levator aponeurosis
Lockwood ligament	<ul style="list-style-type: none"> • Function <ul style="list-style-type: none"> – Forms a hammock supporting the globe – Prevents downward displacement of the eyeball • Composed of <ul style="list-style-type: none"> – Tenon capsule – Intermuscular septum between the inferior oblique muscle and inferior rectus muscle – Medial and lateral check ligaments • Fuses anteriorly with inferior suspensory ligament of conjunctival fornix • Fuses posteriorly with capsulopalpebral fascia
Medial and lateral check ligaments	<ul style="list-style-type: none"> • Function <ul style="list-style-type: none"> – Limit muscle movement • Composed of <ul style="list-style-type: none"> – Medial or lateral extension of the orbital septum – Medial or lateral extension of the levator aponeurosis – Sheath of rectus muscles (medial or lateral) • Attached to the posterior lacrimal crest (via medial canthal ligament) • Attached to the Whitnall tubercle (via lateral canthal ligament)
Medial canthus	<ul style="list-style-type: none"> • Does not contact the ocular globe; helps to collect tears • Two heads <ul style="list-style-type: none"> – Anterior part: Major <ul style="list-style-type: none"> ◦ Tendonous attachment of orbicularis oculi muscles (pretarsal) ◦ Ligament to tarsal plate – Posterior part: Minor <ul style="list-style-type: none"> ◦ Horner muscle ◦ Tendonous attachment of orbicularis oculi muscle
Lateral canthus	<ul style="list-style-type: none"> • Anchors the tarsus of both lids laterally to the Whitnall tubercle • Contacts the ocular globe and causes tears to flow medially • Consists of <ul style="list-style-type: none"> – Lockwood ligament – Lateral horn of levator aponeurosis – Continuations of the pretarsal and preseptal muscles – Check ligament of the lateral rectus • Two heads: Anterior and posterior (major)

Miscellaneous Structures

Annulus of Zinn	<ul style="list-style-type: none">• Ring of fibrous tissue surrounding the optic nerve at the apex• Origin of all extraocular muscles at the apex of the orbit except inferior oblique muscle
Tenon capsule	<ul style="list-style-type: none">• Fibrous tissue (connective tissue network) that surrounds the eyeball and extraocular muscles• Posterior extension of the sclera or the anterior extension of the dura• Provides support to the globe• Allows coordinated movements among all the orbital contents
Orbit	<ul style="list-style-type: none">• Seven bones: zygoma, maxilla, sphenoid, ethmoid, lacrimal, palatine, and frontal• Volume: 30 cc• Weight: 7.5 g• Dissection – Safe 25 to 30 mm from the rim – Medial orbital wall<ul style="list-style-type: none">◦ Anterior ethmoid artery is 24 mm from the anterior lacrimal crest◦ Posterior ethmoid artery is 36 mm from the anterior lacrimal crest◦ Optic canal is 42 mm from the anterior lacrimal crest– Superior orbital wall<ul style="list-style-type: none">◦ Optic canal is 45 mm from the supraorbital notch◦ Supraorbital nerve is approximately 27 mm from facial midline

Physical Findings

- Subconjunctival hemorrhage
- Periorbital ecchymosis
- Diplopia
- Bony steps
- Paresthesia
- Limited ocular movement (positive forced duction test = entrapment of inferior rectus muscle)
- Globe injury in 30% of all orbital fractures (corneal abrasion, globe rupture, hyphema)
- Retrobulbar hemorrhage

Treatment

Types of fracture	<ul style="list-style-type: none">• Medial orbital wall: Most common through lamina papyracea• Orbital floor: Medial to the infraorbital nerve—thinnest part of the floor• Lateral orbital wall: Associated with ZMC fracture• Orbital roof: Associated with NOE and/or frontal sinus fracture
When to observe	<ul style="list-style-type: none">• Slightly or nondisplaced internal orbital fracture without disturbance of eye mobility• Orbital fracture of the only functioning eye• Hyphema, retinal tear, globe perforation• Medically unstable patient

(Treatment cont)

When to consider surgery	<ul style="list-style-type: none"> • A significant increase in the orbital volume as seen on CT scan • Globe malposition (ie, enophthalmos, hypophthalmos) • Nonresolving diplopia • Comminuted orbital rim by CT scan • > 50% floor disruption by CT scan
Timing of treatment	<ul style="list-style-type: none"> • Immediate <ul style="list-style-type: none"> – A positive forced duction test (especially in a child, white-eyed blowout fracture; see page 190) <ul style="list-style-type: none"> ◦ Indicates a trapdoor-type fracture that has incarcerated the inferior rectus muscle, which can result in necrosis of the muscle – Globe displacement into maxillary sinus – Orbital fracture associated with optic nerve compression should be treated within 2 hours • Delayed—within 2 weeks <ul style="list-style-type: none"> – Most adult fractures fall into this category – Allow edema to resolve; easier surgical reduction
Nonsurgical treatment	<ul style="list-style-type: none"> ◦ Antibiotics: Routine use is not recommended ◦ Swelling: Steroid (dexamethasone) <ul style="list-style-type: none"> – Decreases swelling, which can help later surgery, if indicated ◦ Sinus precautions: Limit nose blowing <ul style="list-style-type: none"> – Prevents transfer of bacteria into the periorbital tissues and periorbital emphysema ◦ Ocular pressure <ul style="list-style-type: none"> – 12–22 mm Hg: Normal – 22–30 mm Hg: Medical therapy (acetazolamide or topical timolol) – > 30 mm Hg: Surgical decompression

Incision Designs

Subciliary incision	<ul style="list-style-type: none"> • Increased risk of ectropion (14%), good access to orbital floor • Difficult to reach medial orbital wall
Transconjunctival incision	<ul style="list-style-type: none"> • Most cosmetic approach • Decreased risk of entropion • Dissection can be done preseptally or postseptally • Lateral canthotomy can be done to increase exposure <ul style="list-style-type: none"> – Important to reattach the ligament to the lateral orbit inside and behind the rim at the end of the procedure
Infraorbital incision	<ul style="list-style-type: none"> • Excellent floor exposure, difficult medial and lateral wall exposure • Least cosmetic approach except in the elderly
Transmaxillary/transnasal incision	<ul style="list-style-type: none"> • Performed using endoscopic assistance • Technique sensitive • Indicated for isolated orbital floor fracture only • Mesh is usually placed without screw fixation

(Incision Designs cont)

Transcaruncle incision	<ul style="list-style-type: none"> • Medial extension of transconjunctival incision • Used to expose the medial wall
Upper lid skin crease incision (upper blepharoplasty)	Preferred access to the lateral wall; allows fixation at the zygomaticofrontal and zygomaticosphenoid sutures
Lateral brow incision	Good access to the zygomaticofrontal suture but leaves a noticeable scar
Coronal incision	<ul style="list-style-type: none"> • Can be elevated below the superior orbital rim for access to the orbital roof and medial and lateral walls • Care must be used to avoid injury to the supraorbital neurovascular bundles

Biomaterials for Orbital Reconstruction

- Titanium mesh
- Autogenous bone graft
- Porous polyethylene (PPE)
- Composite of PPE and titanium mesh

Complications

Orbital hematoma	<ul style="list-style-type: none"> • Signs and symptoms <ul style="list-style-type: none"> – Proptosis – Ecchymosis – Afferent pupil defect – Hard globe – Superior orbital fissure syndrome – Orbital apex syndrome • Treatment <ul style="list-style-type: none"> – Lateral canthotomy (immediately) – Intravenous acetazolamide (500 mg) – Intravenous mannitol (0.5 g/kg)
Ectropion	<ul style="list-style-type: none"> • Eversion of the eyelid • Risk factors <ul style="list-style-type: none"> – Subciliary incision (14% versus 3% with transconjunctival incision) – Elderly patients – Lateral canthotomy • Signs and symptoms <ul style="list-style-type: none"> – Epiphora – Ocular irritation – Impaired cosmesis • Treatment <ul style="list-style-type: none"> – Artificial tears – Tarsal stripping, Frost suture

(Complications cont)

Entropion	<ul style="list-style-type: none"> • Inversion of the eyelid • More common when transconjunctival incision is used • Signs and symptoms <ul style="list-style-type: none"> – Redness – Epiphora – Decreased vision due to corneal damage – Sensitivity to light and wind • Treatment <ul style="list-style-type: none"> – Temporizing Quickert-Rathbun sutures (passing a gut suture from the inferior fornix anteriorly toward the lashes) – Severe cases may require oral mucosal graft
Persistent diplopia	<ul style="list-style-type: none"> • Usually occurs in extreme gaze: No treatment is usually needed • Diplopia in primary gaze: Treat with corrective prism and eye muscle surgery
Posttraumatic enophthalmos	<ul style="list-style-type: none"> • Every 1 cc increase in orbital volume produces 0.9 mm increase in enophthalmos • Causes – Improper orbital volume restoration (usually due to displacement of medial, lateral, and posterior floor) <ul style="list-style-type: none"> – Orbital fat atrophy – Dislocation of the trochlea – Cicatricial contraction of the retrobulbar tissues

Special Considerations

Panfacial Fractures

- Fractures include the bones of the upper, middle, and lower face
- Loss of cranial relationships with both the maxilla and the mandible
- Observed in 15% of patients with injuries
- Face tends to assume a spherical appearance
- Face seems wide and elongated and loses projection in the nasal region
- Vertical buttresses: Nasomaxillary, zygomaticomaxillary, and pterygomaxillary
- Horizontal buttresses
 - Frontal bar (supraorbital rim and glabellar region)
 - Infraorbital rim
 - Zygomatic arch
 - Basal segment of the mandible

Approaches

- **Bottom-to-top reconstruction:** The mandible can serve as a foundation and reference for the midface reconstruction
- **Top-to-bottom reconstruction:** Fractured bones near the cranial base are found in larger segments, which makes it easier to achieve anatomical reduction
- **Zone-type approach (Manson and Markowitz):** Dividing the face into anatomical units, the upper and lower face are divided at the Le Fort I level, and each of these halves are divided into two facial units; repair begins with the assembly of a single specific area of the facial skeleton, followed by subsequent integration of that area with the other units
- **Periphery-to-center approach (Gruss and Philips):** By stressing the importance of setting the zygomatic arch, this approach produces an intact outer facial frame upon which the inner frame can be built

Preoperative considerations

- Surgical splint—Indications
 - Palatal split fracture is present
 - Unstable occlusion or comminuted mandibular and/or maxillary fractures
- Intubation
 - Tracheostomy
 - Submental intubation
- Condyle fracture—Indications for open reduction (see page 175)

Common approach sequences

Mandible can be reconstructed	Mandible cannot be reconstructed
<ol style="list-style-type: none">1. Place arch bars on the teeth2. Reduce and stabilize all mandibular fractures; this requires ORIF of any condylar fractures3. Place MMF; a splint is required if a palatal fracture is present4. Expose all midface and upper face fractures5. Treat the frontal sinus as needed6. Treat the NOE fractures; medialize the frontal processes of the maxilla; bone grafting the nose should be delayed until just before closure7. If there are ZMC fractures that are separate from the Le Fort fractures, reduce and stabilize the zygomas; this may require ORIF of the arches8. Rotate the maxillomandibular complex upward until bone contacts at the Le Fort I level; plate across the Le Fort I level9. In the case of a monoblock Le Fort III fracture, mobilize it, place the patient into MMF (if not already), and rotate the maxillomandibular complex upward until bone contacts at the frontozygomatic region; plate across the frontozygomatic fractures10. Remove the MMF and check the occlusion11. Reconstruct the internal orbits12. Reconstruct the dorsum of the nose with bone graft as needed13. Close soft tissues	<ol style="list-style-type: none">1. Place arch bars on the teeth2. Expose all midface and upper face fractures3. Treat the frontal sinus as needed4. Treat the NOE fractures, taking care to medialize the frontal processes of the maxilla; bone grafting the nose should be delayed until just before closure5. If there are ZMC fractures that are separate from the Le Fort fractures, reduce and stabilize the zygomas; this may require ORIF of the arches6. Reduce the maxilla at the Le Fort I level until the best-fit bone contacts are established; plate across the Le Fort I level7. In the case of a monoblock Le Fort III fracture, reduce the Le Fort III complex by best fit to the surrounding bones, and plate across the frontozygomatic fractures8. Place the patient into MMF9. Open, reduce, and stabilize those mandibular fractures that are possible to treat10. Close soft tissues11. Reconstruct the internal orbits12. Reconstruct the dorsum of the nose with bone graft as needed

Pediatric Trauma Special Considerations

Epidemiology

- Most common age group: 6 to 12 years
- Boys > girls
- Causes: Motor vehicle–related accidents > falls > sports injuries > interpersonal violence
- Location
 - Toddlers and infants more likely to experience midfacial and cranial injuries
 - Adolescents associated with mandibular fractures
 - Dentoalveolar and nasal fractures should be the most common fractures but are usually underreported
- Frequency: Mandibular fractures (condyle fractures > symphyseal region > body > angle of the mandible) > orbital fractures > dentoalveolar fractures > midface fractures > nasal fractures > complex fractures > cranial fractures

Growth	<ul style="list-style-type: none">• Maxilla: Forward and downward• Mandible body: Forward and downward• Mandibular rami and condyles: Upward and backward• Skeletal maturity of maxilla and mandible: Between 14 and 16 years of age in females and 16 and 18 years of age in males• Skeletal maturity of orbit: Between 5 and 7 years of age
Mixed dentition	<ul style="list-style-type: none">• Must be considered due to erupting tooth buds, especially with ORIF; primary teeth can be challenging for placement of MMF devices• Increased interdental space among primary teeth makes it difficult for arch bar placement
Closed reduction	<ul style="list-style-type: none">• Most pediatric facial fractures can be treated with closed reduction (Risdon cable)• A lingual splint for mandibular fractures is also recommended• Can use ORIF, but some advocate removing hardware once healed if the child is still growing
Physiotherapy	Range of motion exercises are very important, especially with closed reduction and MMF
Resorbable hardware	<ul style="list-style-type: none">• Bulky and oversized• Risk of damaging tooth buds• Sterile abscess possible from degradation

Differences between pediatric and adult fractures

Fracture types	Pediatric differences
Frontal bone and superior orbital fractures	<ul style="list-style-type: none"> • More common than in adults due to prominent features in the upper third of the face during early childhood • Displacement (full thickness of bone) of the anterior cranial vault and superior orbital rim requires surgical treatment
Frontal sinus and frontobasilar injuries	<ul style="list-style-type: none"> • Frontal sinus starts to develop around 1 to 2 years of age • Not radiographically visible until 6 years old • Osteomyelitis of the skull is less common in children
Orbital fractures	<p>White-eyed blowout fracture</p> <ul style="list-style-type: none"> • Orbital floor fracture in young patients with little or no clinical evidence of soft tissue trauma; however, persistent diplopia with restriction of vertical gaze is appreciated • Due to entrapment of orbital tissue in the orbital floor fracture; bone is softer and more flexible than adult bone • Treatment requires immediate surgery; delayed treatment (> a week) can lead to muscle necrosis and residual motility deficit and diplopia
ZMC fractures	<ul style="list-style-type: none"> • Caution necessary in placing internal fixation in the ZM buttress because of unerupted tooth buds • Limit wide stripping of the soft tissue in the immature skeleton to avoid periosteal scarring and inhibition of future growth
Nasal fractures	<ul style="list-style-type: none"> • Cartilages of a child's nose bend easily, and the nose does not project as much, which leads to a decreased incidence of nasal fractures • Most reductions can be performed with closed techniques • Open techniques should be as conservative as possible with preservation and not resection of cartilage to prevent long-term growth disturbances
Midface fractures	<ul style="list-style-type: none"> • Midface fracture is less common because of a prominent forehead and underdeveloped maxillary sinuses • Caution necessary in placing internal fixation because of tooth buds
Mandibular fractures	<ul style="list-style-type: none"> • Closed reduction is recommended • If open reduction is needed to gain proper alignment <ul style="list-style-type: none"> – Arch bar with transoral monocortical fixation on the inferior border; guiding elastics postoperatively – Lingual splint
Condyle fractures	<ul style="list-style-type: none"> • 10% of patients with dentofacial deformity have evidence of previously undiagnosed condyle fractures • Greater propensity for fracture through the condylar head rather than the neck because of a thick and short condylar neck • Long-term follow-up is needed to observe for ankylosis, especially with condyle fracture with lateral or superolateral displacement because of close contact with body of the zygoma

Firearm Injuries to Head and Neck

Epidemiology

- 115,000 cases per year
- 10% of cases are head and neck related: Midface > mandible
- 30% of cases are fatal
- Causes: 51% attempted suicide; 14% assault; 12% accidental injury; 23% unknown
- Handgun > shotgun > rifles

Definitions

- **Penetrating wounds:** Projectile enters the victim but does not exit
- **Perforating wounds:** Projectile enters and exits the victim; avulsive; substantial loss of tissue occurs
- **Permanent cavity:** Tissue destruction as a result of direct passage of the projectile and its fragments
- **Temporary cavity:** Transient radial deformation of tissues adjacent to the permanent cavity as the projectile passes; in elastic soft tissues, the temporary cavity may be larger than the permanent cavity; tissue injury is in a manner similar to that of blunt trauma

Wound characteristics	
Entry wound	<ul style="list-style-type: none">• Usually smaller than the exit wound• Consists of wound, tissue abrasion, burn, and tattooing
Exit wound	<ul style="list-style-type: none">• Not all gunshot wounds will have exit wounds; on occasion there will be multiple exit wounds due to fragmentation of bone or the bullet• Generally, the exit wound is larger and has ragged edges
Internal wound	<ul style="list-style-type: none">• Medium-velocity bullets inflict damage primarily by injuring tissue that the bullet contacts• High-velocity bullets inflict damage by tissue contact and transfer of kinetic energy

Differences Between Weapons

	Handgun	Rifle	Shotgun
Velocity	Low velocity: < 2,000 ft/s	High velocity: > 2,000 ft/s	Low velocity: < 2,000 ft/s
Ammunition	Bullet	Full-metal jacket	Cartridge: pellets (3 types)

(Differences Between Weapons cont)

	Handgun	Rifle	Shotgun
Wound	<ul style="list-style-type: none">Entrance wound is smaller than the diameter of the bulletCircular or oblong wound tracksUsually no exit wound or slit-shaped or stellate exit wound	<ul style="list-style-type: none">Irregular entrance wound marginsTemporary cavity may be as much as eight times the diameter of the projectileStellate exit wound with tissue avulsion	<p>Type 1</p> <ul style="list-style-type: none">> 7 yards awayPellet scatter within area of 25 cm²Multiple pellet holesMild head and neck injury <p>Type 2</p> <ul style="list-style-type: none">3 to 7 yards awayPellet scatter within area between 10 and 25 cm²Moderate head and neck injuryEntrance wound with some satellite exit wounds <p>Type 3</p> <ul style="list-style-type: none">< 3 yards awayPellet scatter within < 10 cm²Single “explosive” entrance woundSevere head and neck injury

Management

Firearm injuries should be managed in three different phases.

Initial phase (immediate)

- ATLS
- Clinical evaluation
- Airway management (tracheotomy or intubation)
- Bleeding control
- Initial soft tissue reapproximation

Second phase (days 1 to 7)

- Aggressive debridement
 - Multiple attempts are needed; most necrotic tissue does not declare itself until 2 or 3 days later
 - Burned tissue is managed with sulfadiazine cream 1%
 - Bone fragments are removed, and the wound is irrigated
 - Nerve repair via primary neurorrhaphy if proximal and distal ends can be found; no nerve grafting should be done until third phase of surgery
- Bone fractures
 - Closed reduction if comminuted fractures or questionable soft tissue vitality
 - Open reduction and internal fixation if possible
- Antibiotics
 - A critical level of bacteria (10⁵ bacteria per gram of tissue) is needed to cause infection, and it may be reached as soon as 6 hours after injury
 - Cephalosporin or extended-spectrum penicillin

Third phase (day 7 and after)

- Reconstruction should wait until adverse conditions are resolved
 - Edema, microvascular damage, and venous congestion have subsided
 - White blood cell count is normalized
 - Body temperature is normalized

- Nonvascularized bone graft
 - Can be successful if the defect is < 8 cm
 - Must be surrounded by well-vascularized soft tissue
- Soft tissue: Pedicle versus free flap based on size, location, and quality of injury
- Nerve grafting can be done with sural, greater auricular, or medial antebrachial cutaneous nerve

Management of Complications

Airway	<ul style="list-style-type: none"> • Evaluate the amount of edema in the larynx • Continuous pulse oximetry and care in an intensive care unit should be considered in the polytraumatized patient • Bilateral condylar fractures should be rapidly assessed and stabilized to avoid the possibility of airway impingement, especially in the overweight patient
Hemodynamic	<ul style="list-style-type: none"> • A postoperative complete blood count as well as hemoglobin and hematocrit levels are important to evaluate blood loss and systemic response to surgical trauma • Angiography and embolization is recommended in the acute setting if the bleeding cannot be stopped by local measures
Ophthalmologic	<ul style="list-style-type: none"> • Cosmetic problems are common with orbital approaches: Ectropion and entropion, as well as hypophthalmos and enophthalmos, can occur after orbital trauma that is not adequately treated; restoring the orbital volume can be challenging, and the use of three-dimensional (3D) technology is encouraged for both navigation and placement of reconstruction materials • Nasolacrimal duct injuries or obstruction will usually manifest as epiphora; it can be diagnosed with a fluorescein test using the Jones technique and usually treated with a dacryocystorhinostomy
Open fractures	Antibiotic use is recommended
Hardware-related	The exposed hardware initially can be preserved provided that adequate irrigation and cleaning of the site is done. If loose hardware is identified it should be removed or replaced
Animal bites	Such bites cause an infection in approximately 20% of cases. The most common bacteria include <i>Pasteurella</i> or <i>Capnocytophaga</i> species. Adequate cleaning and irrigation, in conjunction with antibiotics, should be the initial treatment of choice (see page 171)
Osteomyelitis	Osteomyelitis may require the use of systemic antibiotics via a peripherally inserted central line. It is preferable to consult and manage these cases with the infectious disease service. Conditions like meningitis and cavernous sinus thrombosis (CST) could occur and should be identified rapidly. CST presents with dramatic periorbital edema, proptosis, ophthalmoplegia, and chemosis, and mortality can be as high as 30%
Nonunion	Nonunion can occur after any ORIF of a fracture. It is usually the consequence of infection, hardware failure, or micromotion. If nonunion is encountered, a second operation is needed to reestablish continuity; thorough debridement of the area and use of new hardware are required. Often malocclusion can occur simultaneously and should be corrected. An autogenous bone graft is recommended in most cases

Recommended Readings

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Pathology

Din Lam and Eric R. Carlson

- ▶ Odontogenic Tumors
- ▶ Nonodontogenic Tumors
- ▶ Odontogenic Cysts
- ▶ Non-neoplastic Salivary Gland Diseases
- ▶ Oral Squamous Cell Carcinoma
- ▶ Cutaneous Lesions
- ▶ Neck Masses
- ▶ Vesiculobullous Diseases
- ▶ Vascular Anomalies
- ▶ Osteomyelitis
- ▶ Osteoradionecrosis
- ▶ Medication-Related Osteonecrosis of the Jaws (MRONJ)
- ▶ Pediatric Pathology
- ▶ Pathology Correlations

Odontogenic Tumors

	Epithelial	Mesenchymal	Mixed
Benign	<ul style="list-style-type: none"> • Ameloblastoma • Adenomatoid odontogenic tumor (AOT) • Pindborg tumor, also known as <i>calcifying epithelial odontogenic tumor (CEOT)</i> • Squamous odontogenic tumor (SOT) • Keratocystic odontogenic tumor (KCOT),* previously known as <i>odontogenic keratocyst (OKC)</i> 	<ul style="list-style-type: none"> • Odontogenic myxoma and fibromyxoma • Odontogenic fibroma • Cementoblastoma • Central granular cell odontogenic tumor (CGCOT) 	<ul style="list-style-type: none"> • Ameloblastic fibroma • Ameloblastic fibro-odontoma (AFO) • Odontoma • Calcifying cystic odontogenic tumor (Gorlin cyst)[†]
Malignant	<ul style="list-style-type: none"> • Primary intraosseous odontogenic carcinoma (PIOC) • Ameloblastic carcinoma • Metastasizing (malignant) ameloblastoma • Clear cell odontogenic tumor • Ghost cell odontogenic carcinoma 		<ul style="list-style-type: none"> • Ameloblastic fibrosarcoma

*According to World Health Organization (WHO) classification of odontogenic tumors. Most clinicians feel the WHO evidence is insufficient for its renaming. The detail of this pathology can be found in the section of odontogenic cysts.

[†]According to WHO classification. Notwithstanding, some of these neoplasms are justifiably classified as cysts, as had been historically recognized.

Origins of Odontogenic Tumors

- Prefunctional dental lamina (odontogenic epithelium with ability to produce a tooth)
 - Abundant distal to the mandibular third molars
- Postfunctional dental lamina
 - Epithelial rests of Serres: Epithelial remnants in fibrous gingival tissue
 - Rests of Malassez: Epithelial remnants (Hertwig root sheath) in periodontal ligament space; responsible for most periapical cysts but not neoplasms (except SOT)
 - Reduced enamel organ epithelium: Covers the enamel surface until tooth eruption; fluid accumulation between this layer and the crown leads to a dentigerous or follicular cyst
- Basal cell layer of the gingival epithelium
- Dental papilla: Origin of the dental pulp, which has the potential to be induced to produce odontoblasts and synthesize dentin and/or dentinoid material
- Dental follicle
- Periodontal ligament

Odontogenic Tumors: Epithelial Origin

Ameloblastoma

- **Origin:** Rests of Serres
- **Histology:** Reverse polarity of nuclei pathognomonic
- Six histologic subtypes: Follicular, plexiform (most common), granular, acanthomatous, desmoplastic, basal cell
 - Follicular: Palisaded ameloblastlike cells and inner zone of triangular-shaped cells resembling stellate reticulum in bell stage (Fig 7-1)
 - Plexiform: Epithelium that proliferates in a “fishnet pattern” (Fig 7-2)
 - Granular: Younger patients, historically thought to be more clinically aggressive
- **Radiograph:** Radiolucency with multiloculated, soap-bubble appearance (Fig 7-3) except for desmoplastic subtype, which is radioopaque due to collagenized stroma; root resorption is common
- **Clinical presentation:** Solid and/or cystic, buccolingual expansion, peripheral painless warty appearance
- **Location:** Posterior mandible is desmoplastic; anterior maxilla is peripheral

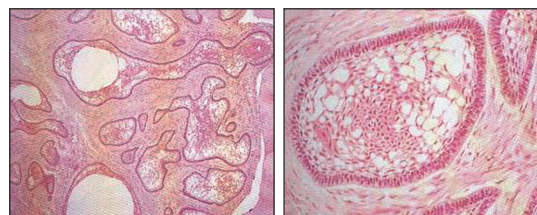


Fig 7-1 Follicular ameloblastoma.

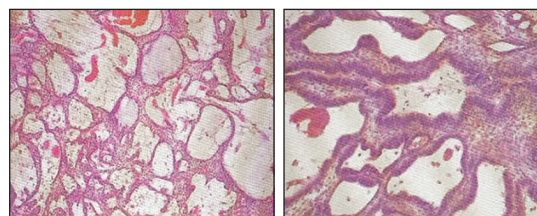


Fig 7-2 Plexiform ameloblastoma.



Fig 7-3 Ameloblastoma soap-bubble radiolucency.

Types*	Decade	Treatment	Recurrence
Solid or multicystic	4th	<ul style="list-style-type: none"> • Resection with 1 cm margin • One anatomical barrier 	<ul style="list-style-type: none"> • Enucleation: 60% to 80% • Resection: 15%
Unicystic subtypes <ol style="list-style-type: none"> 1. Luminal with simple odontogenic epithelial lining 2. Intraluminal plexiform proliferation of the epithelial lining 3. Mural with epithelium invading connective tissue 	3rd	<ul style="list-style-type: none"> • Unicystic: Less aggressive; enucleation and simple curettage +/- peripheral ostectomy or liquid nitrogen cryotherapy • Mural: Consider resection with 1-cm margin 	No recurrence
Peripheral	6th	Excision	No recurrence

*Unicystic versus multicystic: Many appear unilocular on image but are multilocular intraoperatively.

Adenomatoid odontogenic tumor (AOT)

- $\frac{2}{3}$ tumor: $\frac{2}{3}$ associated with canine; $\frac{2}{3}$ females; in the 2nd and 3rd decades of life
- **Origin:** Hertwig epithelial root sheath
- **Histology:** Columnar epithelial in ductlike pattern (Fig 7-4)
- **Radiograph:** Pear-shaped radiolucency with speckled opaque foci (Fig 7-5)
- **Clinical presentation:** Asymptomatic expansion
- **Treatment:** Enucleation and curettage

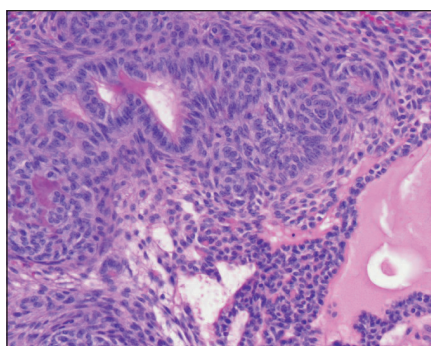


Fig 7-4 AOT ductlike pattern.



Fig 7-5 AOT pear-shaped radiolucency.

Pindborg tumor or calcifying epithelial odontogenic tumor (CEOT)

- **Origin:** Stratum intermedium
- **Age:** between 13 and 80 years
- **Histology:** Liesegang rings, psammomalike calcifications (Fig 7-6); if clear cells present, increased aggressiveness and cortical perforation; amyloid is a characteristic finding
- **Radiograph:** Well-defined mixed radiolucent/radiopaque; unilocular or multilocular presentation; snowflake pattern (Fig 7-7)
- **Location:** Mandibular premolar region
- **Clinical presentation:** Asymptomatic
- **Treatment:** Resection with 1-cm margin; 14% to 20% recurrence with enucleation

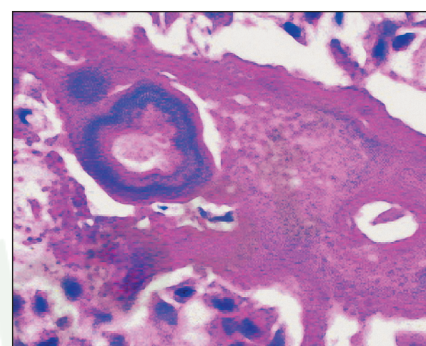


Fig 7-6 Liesegang rings characteristic of a Pindborg tumor.

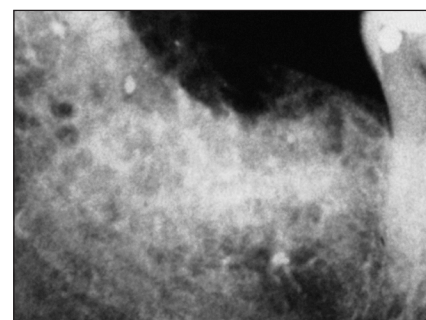


Fig 7-7 Pindborg tumor snowflake pattern.

Squamous odontogenic tumor (SOT)

- **Origin:** Rests of Malassez
 - **Histology:** Variable-sized nests of cytologically bland squamous epithelium in a moderately cellular fibrous stroma
- **Age:** Mean age of 40 years; between the 2nd and 7th decades of life
- **Radiograph** (Fig 7-8): Triangular- or pear-shaped radiolucency associated with erupted tooth (similar to AOT except tooth is erupted)
- **Location:** Anterior maxilla and/or posterior mandible



Fig 7-8 SOT triangular radiolucency.

Odontogenic Tumors: Mesenchymal

Odontogenic myxoma

- **Origin:** Dental papilla
- **Histology:** Predominance of stellate and spindle cells surrounded by mucoid material (Fig 7-9)
- **Age:** Between 2nd and 4th decades; young adults between 25 and 35 years
- **Radiograph**
 - Ill-defined radiolucent border; mimics malignancy
 - > 50% are multilocular
 - Similar to ameloblastoma; soap-bubble or honeycomb pattern (Fig 7-10)
- **Location:** Mandible > maxilla; anterior for unilocular; posterior for multilocular
- **Clinical presentation:** Asymptomatic, expansion associated with unerupted tooth; root displacement/or resorption
- **Treatment:** Segmental resection with 1-cm margin; recurrence uncommon

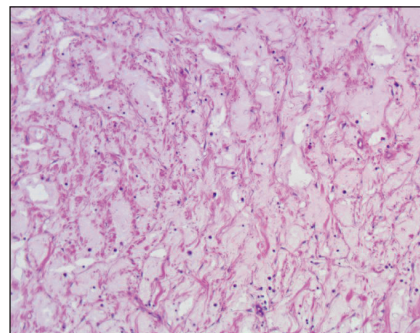


Fig 7-9 Myxoma.



Fig 7-10 Myxoma honeycomb radiolucency.

Odontogenic fibroma

- **Origin:** Periodontal ligament
- **Histology** of two types
 - WHO subtype: Well demarcated or encapsulated; islands and strands of inactive odontogenic epithelium can be found
 - Non-WHO subtype: Delicate fibrous tissue, which may contain various amounts of collagen
- **Age:** Wide range, average of 40 years
- **Radiograph:** Either radiolucent or mixed radiolucent and radiopaque
- **Location:** Anterior maxilla or posterior mandible
- **Clinical presentation:** Painless expansion that may displace and/or resorb tooth roots
- **Treatment:** Enucleation and curettage with no recurrence

Cementoblastoma

- **Origin:** Cementoblasts forming disorganized cementum
- **Histology:** Masses of cementum with prominent basophilic reversal lines and cementoblastic rimming; cementoblasts plump with hyperchromatic nuclei and conspicuous nucleoli, loose fibrovascular stroma with scattered multinucleated osteoclast-type giant cells
- **Age:** Teenage, < 30 years
- **Radiograph**
 - Spherical radiopaque mass encompassing and essentially replacing the apical half of the root
 - Periodontal ligament space surrounds the radiopaque mass, distinguishable feature from hypercementosis (Fig 7-11)
- **Location:** Mandibular molars and premolars (apical half of root)
- **Clinical presentation:** Expansion with deep, dull pain; associated tooth is vital
- **Treatment:** Removal of tooth with associated lesion, no surgical margin is needed; recurrence rate is low

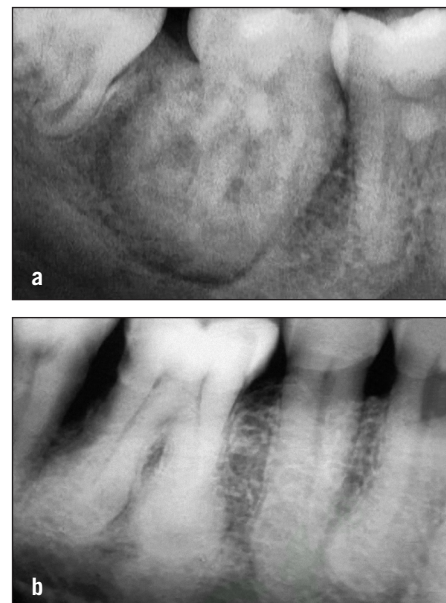


Fig 7-11 Comparison of radiographs of cementoblastoma (a) and hypercementosis (b).

Central granular cell odontogenic tumor (CGCOT)

- **Age:** > 40 years
- **Histology:** Large eosinophilic granular cells and small cords of odontogenic epithelium
- **Radiograph:** Well-defined radiolucency with some opacity
- **Location:** Maxilla and mandible; molar region of both jaws
- **Clinical presentation:** Bony expansion
- **Treatment:** Enucleation and curettage; recurrence is rare

Odontogenic Tumors: Mixed

Ameloblastic fibroma

- **Origin:** Dental lamina
- **Age:** 1st and 2nd decades of life; average age is 14 years
- **Histology:** Islands and narrow cords of odontogenic epithelium in a cellular, myxoid, mesenchymal stroma
- **Radiograph**
 - Unilocular or multilocular radiolucency
 - 50% are associated with an unerupted tooth
- **Location:** Posterior mandible
- **Clinical presentation:** Small tumors usually are asymptomatic
- **Treatment:** Enucleation and curettage; recurrence uncommon

Ameloblastic fibro-odontoma (AFO)

- Hybrid of ameloblastic fibroma and odontoma
- **Origin:** Dental lamina
- **Age:** Between 5 to 12 years with a mean of 10 years
- **Histology:** Soft tissue component is identical to the ameloblastic fibroma, with calcified foci of enamel and dentin matrix

- **Radiograph:** Mixed radiolucent-radiopaque lesion
- **Location:** Premolar and molar region of both jaws
- **Clinical presentation:** Slow growing, asymptomatic, bony expansion
- **Treatment:** Enucleation and curettage; recurrence is rare

Odontoma

- **Age:** 2nd decade; mean is 14 years
- **Types:** Compound and complex
- **Histology:** Compound has dental tissues arranged in toothlike structures; complex is an unstructured mass of dental tissues
- **Radiograph**
 - Early lesion is well-defined radiolucency; late lesion is well-defined radiopacity
 - Compound is toothlike; complex is radiopaque lesion
- **Location:** Compound in anterior maxilla; complex in posterior maxilla and mandible (Fig 7-12)
- **Clinical presentation:** Asymptomatic
- **Treatment:** Enucleation and curettage; recurrence rate is rare

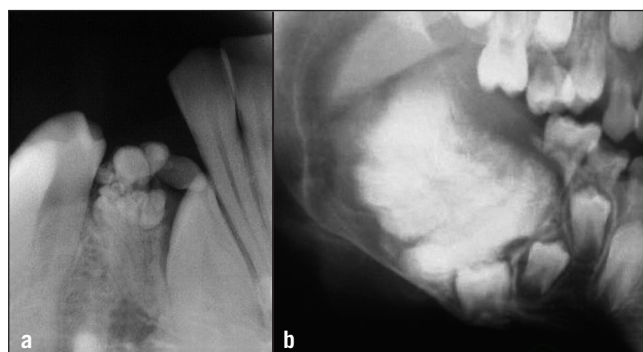


Fig 7-12 Comparison of radiographs showing complex (a) and compound (b) odontomas.

Calcifying cystic odontogenic tumor (Gorlin cyst)

- Controversy exists regarding its behavior: Cyst versus neoplasm
- **Age:** Between the 2nd and 3rd or 6th and 7th decades
- **Origin:** Reduced epithelium or dental lamina remnants
- **Histology:** Suprabasilar epithelium shows stellate reticulum with ghost cell formation (Fig 7-13)
- **Clinical presentation:** Swelling and gingival tenderness; may occur peripherally in 30% of cases
- **Radiograph:** Unilocular or multilocular radiolucency; 10% associated with odontoma
 - Only cyst associated with radiopacity
- **Treatment:** Surgical excision with low recurrence rate
- **Malignant potential:** Rare, odontogenic ghost cell carcinoma

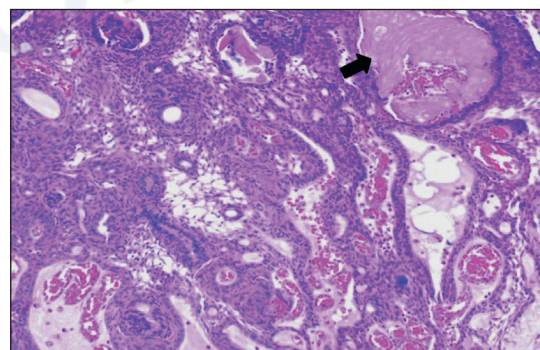


Fig 7-13 Histology of calcifying odontogenic cyst with ghost cell (arrow) formation.

Malignant Odontogenic Tumors

Primary intraosseous odontogenic carcinoma (PIOC)

Waldron and Mustoe classification

- Type 1: PIOC exodontogenic cyst
- Type 2a: Malignant ameloblastoma
- Type 2b: Ameloblastic carcinoma arising de novo, exameloblastoma, or exodontogenic cyst
- Type 3: PIOC arising de novo
 - Keratinizing type
 - Nonkeratinizing type
- Type 4: Intraosseous mucoepidermoid carcinoma

- **Diagnostic criteria**
 - Histologic evidence of squamous cell carcinoma
 - Absence of ulcer formation on the overlying mucosa
 - Absence of a distant primary tumor at the time of diagnosis and at least 6 months during the follow-up period
- **Age:** Average 57 years
- **Location:** Posterior mandible
- **Clinical presentation**
 - Asymptomatic or painful swelling, paresthesia, and loosening of teeth
 - Locally aggressive, rare metastasis to the regional lymph nodes
- **Radiograph:** Variable but most common is an irregular and poorly defined radiolucency
- **Treatment:** Treat as oral squamous cell carcinoma; segmental resection with 1-cm margin, neck dissection, radiation therapy as needed based on multidisciplinary discussion
- **Prognosis:** 30% to 40% 5-year survival rate

	Metastasizing (malignant) ameloblastoma	Ameloblastic carcinoma
Incidence	< 1% of ameloblastomas will develop malignancy	
Histology	Benign ameloblastoma in both the primary and secondary deposits	Malignant tumor in recurrence and any metastases
Metastasis	Lung > cervical lymph nodes	Uncommon
Presentation	Similar to benign solid or multicystic ameloblastoma with more ill-defined or destructive features	
Treatment	Dependent on tissue involved	Resection with 1.0-cm margin
Prognosis	50% mortality rate; poor prognosis	

Clear cell odontogenic carcinoma

- **Age:** > 50 years
- **Histology**
 - Nests of epithelial cells with a clear cytoplasm separated by strands of hyalinized connective tissue
 - Need to rule out other clear cell tumors: calcifying epithelial odontogenic tumor, mucoepidermoid carcinoma, and renal cell carcinoma

- **Radiograph:** Unilocular or multilocular radiolucency with ill-defined margins
- **Location:** Both jaws
- **Clinical presentation:** Expansile tumor with possible paresthesia; metastasizes to lung or cervical lymph nodes
- **Treatment:** Resection with 1.0- to 1.5-cm margins

Ghost cell odontogenic carcinoma

- Malignant form of CEOT, solid type
- Can occur de novo or arise from CEOT
- **Age:** Adolescence to middle age
- **Radiograph:** Unilocular or multilocular radiolucency with ill-defined margins
- **Location:** Posterior maxilla
- **Clinical presentation:** Painful expansion of the jaw with possible paresthesia in the mandible
- Need to rule out other ghost cell tumors: Craniopharyngioma, ameloblastic fibro-odontoma, calcifying odontogenic cyst
- **Treatment:** Resection with 1.0-cm margins

Ameloblastic fibrosarcoma

- Malignant form of ameloblastic fibroma (AF)
- Can occur de novo or arise from AF
- **Age:** Mean of 26 years
- **Histology:** Epithelial component of this tumor appears histologically benign; mesenchymal portion is highly cellular with mitotic figures
- **Radiograph:** Ill-defined, destructive, radiolucent lesion
- **Location:** Mandible
- **Clinical presentation:** Painful, rapid swelling of the jaw
- **Treatment:** Resection with 1- to 1.5-cm margins

Odontogenic Tumors: Prognosis

Benign and low recurrence <ul style="list-style-type: none">• AOT• SOT• Cementoblastoma• Odontoma• Central granular cell odontogenic tumor• Ameloblastic fibro-odontoma	Benign and moderate recurrence <ul style="list-style-type: none">• Unicystic ameloblastoma• AF
Benign, high recurrence, and aggressive <ul style="list-style-type: none">• Solid or multicystic ameloblastoma• Calcifying epithelial odontogenic tumor• Odontogenic myxoma	Malignant <ul style="list-style-type: none">• Primary intraosseous odontogenic carcinoma• Metastasizing (malignant) ameloblastoma• Ameloblastic carcinoma• Clear cell odontogenic carcinoma• Ghost cell odontogenic carcinoma• Ameloblastic fibrosarcoma

Nonodontogenic Tumors

Benign	Giant cell lesions	<ul style="list-style-type: none"> • Hyperparathyroidism (Brown tumor) • Cherubism • Aneurysmal bone cyst (ABC) • Central and peripheral giant cell lesions
	Langerhans cell disease	<ul style="list-style-type: none"> • Unifocal (eosinophilic granuloma) • Multifocal unisystem (Hand-Schuller-Christian disease) • Multifocal multisystem (Letterer-Siwe disease)
	Fibro-osseous disease	<ul style="list-style-type: none"> • Cemento-osseous lesions • Fibrous dysplasia • Ossifying fibroma
	Neurogenic tumor	<ul style="list-style-type: none"> • Schwannoma (also known as <i>neurilemmoma</i>) • Neurofibroma • Melanotic neuroectodermal tumor of infancy
	Osteoid osteoma or osteoblastoma	<ul style="list-style-type: none"> • Osteoid osteoma • Osteoblastoma
	Others	<ul style="list-style-type: none"> • Osteoma • Desmoplastic fibroma • Chondroma
Malignant	Bone	<ul style="list-style-type: none"> • Osteosarcoma • Ewing sarcoma • Fibrosarcoma of bone
	Cartilage	Chondrosarcoma
	Hematopoietic	<ul style="list-style-type: none"> • Malignant fibrous histiocytoma • Burkitt lymphoma • Multiple myeloma
	Nerve	Malignant peripheral nerve sheath tumor
	Others	<ul style="list-style-type: none"> • Postradiation sarcoma • Metastatic carcinoma

Benign Nonodontogenic Tumors

Giant cell lesions

- Lesions are poorly related to one another
- Histologically they are indistinguishable under light microscopy

	Types	Clinical presentation	Treatment												
Hyperparathyroidism	<ul style="list-style-type: none">• Primary: Usually from adenoma (parathyroid)• Secondary: Usually from chronic renal disease leading to hypovitaminosis D and lower calcium (Ca^{2+}) reabsorption• Tertiary: Refractory hyperparathyroidism from chronic renal disease or a paraneoplastic syndrome related to squamous cell carcinoma of the oral cavity; resembles primary hyperthyroidism because of parathyroid-related peptide secretion	<ul style="list-style-type: none">• Asymptomatic or “stones, bones, groans”• Osteitis fibrosa cystica<ul style="list-style-type: none">– Primary hyperparathyroidism only– Cystic bone spaces filled with brown fibrous tissue• Renal osteodystrophy<ul style="list-style-type: none">– Bone lesions in secondary and tertiary <table><tr><th></th><th>Ca^{2+}</th><th>PO_4^{3-}</th></tr><tr><td>Primary</td><td>↑</td><td>↓</td></tr><tr><td>Secondary</td><td>↓</td><td>↑</td></tr><tr><td>Tertiary</td><td>↑</td><td>↓</td></tr></table> <p>PO_4^{3-}, phosphate ion.</p> <ul style="list-style-type: none">– Jaw lesions<ul style="list-style-type: none">◦ Enlargement of the jaw◦ Generalized loss of lamina dura◦ Radiograph: “Ground-glass” (Fig 7-14) because of parathyroid-related peptide secretion		Ca^{2+}	PO_4^{3-}	Primary	↑	↓	Secondary	↓	↑	Tertiary	↑	↓	<ul style="list-style-type: none">• Excision of adenoma in primary hyperparathyroidism• Medical treatment in secondary and tertiary hyperparathyroidism• Lesions will regress when the endocrine abnormality is treated• If needed, curettage is treatment of choice
	Ca^{2+}	PO_4^{3-}													
Primary	↑	↓													
Secondary	↓	↑													
Tertiary	↑	↓													



Fig 7-14 Hyperparathyroidism ground-glass radiolucency.

(Giant cell lesions cont)

	Types	Clinical presentation	Treatment
Giant cell lesion	<p>Central giant cell lesion (CGCL; Fig 7-15)</p> <ul style="list-style-type: none"> • Nonaggressive: Giant cell lesion • Aggressive: Giant cell tumor <p>Nonaggressive versus aggressive</p> <p><i>Major criteria</i></p> <ol style="list-style-type: none"> 1. Size is > 5 cm 2. Recurrence after initial treatment <p><i>Minor criteria</i></p> <ol style="list-style-type: none"> 3. Rapid growth 4. Tooth loosening and/or displacement 5. Radiographic evidence of cortical thinning and/or perforation 6. Radiographic evidence of tooth resorption and/or displacement <p>Aggressive tumor diagnosis requirements</p> <ul style="list-style-type: none"> • One major criterion • Three minor criteria <p>Peripheral</p> <ul style="list-style-type: none"> • Soft tissue equivalent of CGCL • Caused by irritation and/or trauma • One of the three P soft tissue lesions: Pyogenic granuloma, peripheral ossifying fibroma, peripheral giant cell lesion 	<ul style="list-style-type: none"> • Age: 10 to 20 years • Aggressive tumor occurs in younger patients (6.3 years versus 9.3 years) • Nonaggressive tumor <ul style="list-style-type: none"> – Asymptomatic • Aggressive tumor <ul style="list-style-type: none"> – Pain – Swelling – Bone perforation – Root resorption • Radiograph: Unilocular or multilocular radiolucency • Crosses midline • Peripheral <ul style="list-style-type: none"> – 50 to 60 years – Red-blue pedunculated lesion – Mandibular gingiva – < 2 cm in size – Hemorrhage and/or ulceration – Destruction of the underlying alveolar bone; “cupping” or “saucerization” 	<p>Nonaggressive</p> <ul style="list-style-type: none"> • Enucleation and curettage <p>Aggressive</p> <ul style="list-style-type: none"> • En bloc resection (1-cm margin) • Enucleation and pharmacologic therapy <ol style="list-style-type: none"> 1. Intralesional steroid: Anti-angiogenic effect 2. Calcitonin therapy: Inhibits bone resorption 3. Interferon therapy: Both prior effects and stimulation of osteoblast activity <p>Peripheral</p> <ul style="list-style-type: none"> • Surgical excision down to bone • 10% recurrence rate



Fig 7-15 Central giant cell granuloma histology (a), radiograph (b), and clinical specimen (c). (Parts b and c reprinted with permission from Dunfee BL, Sakai O, Pistey R, Gohel A. Radiologic and pathologic characteristics of benign and malignant lesions of the mandible. Radiographics 2006;26:1751–1768.)

(Giant cell lesions cont)

	Types	Clinical presentation	Treatment
Cherubism <ul style="list-style-type: none"> Autosomal dominant Mutation in <i>SH3BP2</i> in chromosome 4p16 Histology: Peri-vascular cuffing of collagen is characteristic but frequently absent 	<ul style="list-style-type: none"> Type 1: Bilateral mandibular rami Type 2: Entire mandible except condyles Type 3: Maxilla and mandible 	<ul style="list-style-type: none"> 2 to 5 years Painless bilateral swelling Nasal obstruction (secondary to middle concha hypertrophy) Multiple radiolucencies Associated with – <ul style="list-style-type: none"> Ramon syndrome Jaffe-Campanacci syndrome Noonan-like 	Observation: Most will resolve spontaneously after puberty
Aneurysmal bone cyst (ABC)		<ul style="list-style-type: none"> Age: Mean of 20 years Painful bony expansion; “blowout” contour Paresthesia Unilocular or multilocular radiolucency with cortical thinning “Blood-soaked” sponge appearance when lesion entered 	Enucleation and curettage

Langerhans cell histiocytosis (LCH)

- Langerhans cells are dendritic mononuclear cells found in epidermis, mucosa, and lymph nodes
- Traditional classification provides limited help in treatment planning
- LCH can occur anywhere in the body; pulmonary LCH is the most common but not related to head and neck LCH
- Diagnosis of Birbeck granules (Fig 7-16) under electron microscopy is gold standard but seldom used
- Diagnosis with immunohistochemical staining: Markers CD1a and S100 (Fig 7-17)
- Most common symptoms: Local bone pain and loose teeth
- Radiographic analysis: “Floating teeth in the air” on panoramic radiograph (Fig 7-18)
- Skeletal survey as needed to search for other skeletal lesions



Fig 7-16 Birbeck granules characteristic of LCH.

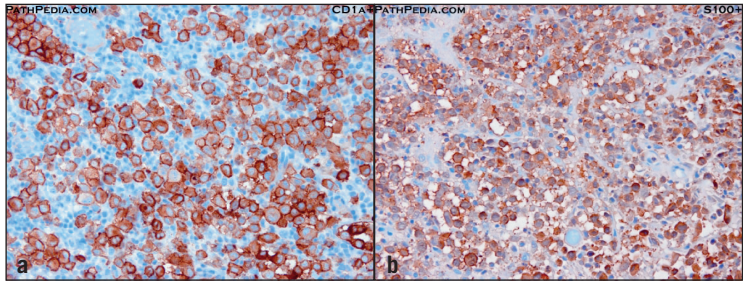


Fig 7-17 CD1a (a) and S100 (b) staining to diagnose LCH. (Photos reprinted with permission from PathPedia.com).



Fig 7-18 Radiograph of LCH with "floating teeth."

	Age	Clinical Presentation	Treatment
Unifocal (eosinophilic granuloma)	Older children and adults	Usually single lesion in bone	<ul style="list-style-type: none">• Observation• Enucleation and curettage• Good prognosis
Multifocal unisystem (Hand-Schuller-Christian)	Young children (< 10 years)	<ul style="list-style-type: none">• Multiple lesions in one organ system• Classical triad includes<ul style="list-style-type: none">– Lesions in the jaws– Exophthalmos– Diabetes insipidus (both related to lesions in the base of the skull involving the pituitary gland)• Few patients have the triad	<ul style="list-style-type: none">• Curettage for accessible lesions• Low-dose radiation for inaccessible lesions (550 to 600 cGy)• Intermediate prognosis
Multifocal multisystem (Letterer-Siwe)	< 2 years	<ul style="list-style-type: none">• Multiple sites and one or more organ systems• Most common locations<ul style="list-style-type: none">– High-risk organs<ul style="list-style-type: none">◦ Bone marrow◦ Lung◦ Liver◦ Spleen– Low-risk organs<ul style="list-style-type: none">◦ Skin◦ Bone◦ Lymph nodes	<ul style="list-style-type: none">• Local excision and radiation for patients with low-risk organ involvement• Chemotherapy with surgery and radiation in patients with high-risk organ involvement• Chemotherapy agents: Vinblastine, prednisone, etoposide• Poor prognosis: 15% mortality

Fibro-osseous lesions

Non-neoplastic intraosseous lesions that replace normal bone with fibrous connective tissue.

	Types	Clinical presentation	Treatment
Cemento-osseous dysplasia (COD) <ul style="list-style-type: none"> • Arises from fibroblasts in the periodontal ligament • All subtypes have same pathology but in different locations • Poor cellularity and vascularity 	Periapical <ul style="list-style-type: none"> • Apices of mandibular anterior teeth (Fig 7-19) Focal <ul style="list-style-type: none"> • Incomplete form of florid COD • Single focus in the alveolar bone of one or both jaws Florid <ul style="list-style-type: none"> • Present in two or more quadrants • Usually in tooth-bearing area bilaterally (Fig 7-20) 	<ul style="list-style-type: none"> • Age: Mean 39 years • Teeth are vital • African American and Asian > white (only in periapical COD and florid COD) • Asymptomatic Florid COD <ul style="list-style-type: none"> • Large lesions can expand the cortices • Associated with simple bone cyst • Can become infected, leading to osteomyelitis Radiograph <ul style="list-style-type: none"> • Early: Well-defined, irregular radiodensity and radiolucency • Late: Well-defined, irregular radiopacity with sclerotic borders 	<ul style="list-style-type: none"> • Observation in asymptomatic patients • Antibiotics in symptomatic patients • Surgical debridement for exposed lesions
Fibrous dysplasia Normal cancellous bone is replaced by fibrous connective tissue	Monostotic <ul style="list-style-type: none"> • Only involves one bone • Craniofacial bone involvement consists of monostotic lesions Polyostotic <ul style="list-style-type: none"> • More than one bone involved Polyostotic with endocrinopathies <ul style="list-style-type: none"> • McCune-Albright syndrome triad <ul style="list-style-type: none"> – Polyostotic fibrous dysplasia – Precocious puberty – Unilateral café au lait spots (irregular “coast of Maine” border) 	<ul style="list-style-type: none"> • Age: 2nd decade, younger in syndromic and craniofacial patients • Asymptomatic, painless swelling • Radiograph “ground-glass” appearance (Fig 7-21) 	<ul style="list-style-type: none"> • Observation, self-resolution after puberty • Surgical contouring if <ul style="list-style-type: none"> – Impinging on vital structures – Significant facial disfigurement

(Fibro-osseous lesions cont)

	Types	Clinical presentation	Treatment
Ossifying fibroma (OF) <ul style="list-style-type: none">• Histologically indistinguishable from fibrous dysplasia• Diagnosis based on clinical and radiographic findings• OF is encapsulated until it reaches 2 to 3 cm in size (fibrous dysplasia is not encapsulated)	<ul style="list-style-type: none">• Conventional• Juvenile ossifying fibroma (JOF) (Fig 7-22)<ul style="list-style-type: none">– More aggressive– Two histologic subtypes<ul style="list-style-type: none">◦ Trabecular◦ Psammomatoid	Conventional OF <ul style="list-style-type: none">• Wide age range; mostly in the 20s and 30s• Mandible > maxilla• Painless expansion Radiograph <ul style="list-style-type: none">– Early stage is radiolucent– Intermediate stage is mixed– Late or mature stage is radiopaque JOF <ul style="list-style-type: none">• Average age 15 years• More common in cra-	<ul style="list-style-type: none">• Enucleation and curettage in conventional OF• Resection with 0.5-cm margin for JOF• Trabecular JOF has better prognosis than psammomatoid JOF



Fig 7-19 Periapical COD.

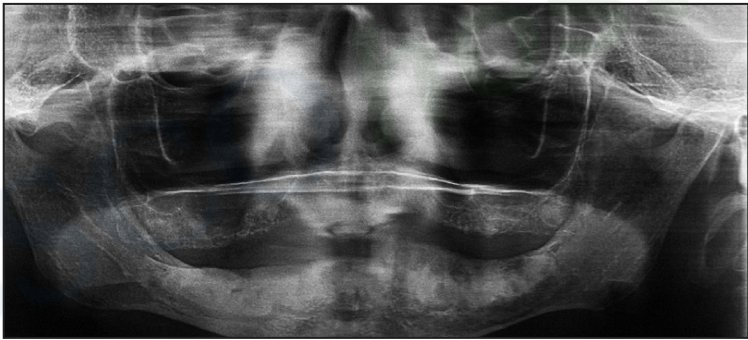


Fig 7-20 Florid COD.



Fig 7-21 Fibrous dysplasia.

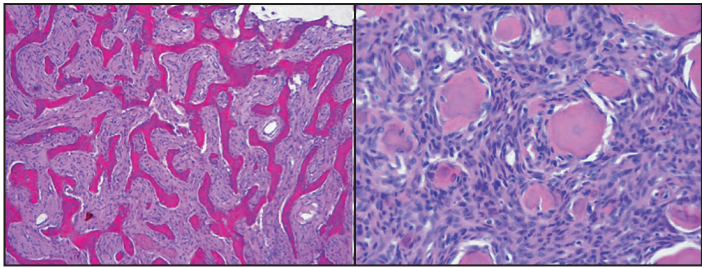


Fig 7-22 JOF histologies.

Neurogenic tumors

	Schwannoma (neurilemmoma)	Neurofibroma
Frequency	Uncommon	Most common type of peripheral nerve neoplasm
Origin	Schwann cells	Mixture of nerve cells (Schwann cells, perineural fibroblasts)
Age	Young and middle age	Young
Location	Tongue and inferior alveolar nerve (IAN)	Skin, tongue, buccal mucosa
Clinical presentation	Asymptomatic; paresthesia if IAN involvement	Asymptomatic
Radiology	Radiolucent (only in intraosseous lesion)	
Histology	<ul style="list-style-type: none"> Pseudo-encapsulated Antoni A and Antoni B forms (Fig 7-23) Verocay bodies (Fig 7-24) 	<ul style="list-style-type: none"> Blends with adjacent tissue Spindle-shaped cells with wavy nuclei Abundant mast cells Subtypes – Dermal: No malignant transformation – Plexiform: Associated with neurofibromatosis and malignant transformation (Fig 7-25)
Treatment	Excision	
Other neurogenic tumors	<p>Ancient schwannoma</p> <ul style="list-style-type: none"> Degenerative changes in some older schwannoma Benign tumor with nuclear atypia Not to be confused with sarcoma <p>Vestibular schwannoma</p> <ul style="list-style-type: none"> Associated with neurofibromatosis type 2 (neurofibromin 2 gene on chromosome 22) 	<p>Associated with neurofibromatosis</p> <ul style="list-style-type: none"> 8 different types Neurofibromatosis type 1 (von Recklinghausen disease) <ul style="list-style-type: none"> Most common (85%) Gene alteration in chromosome 17 (neurofibromin 1 gene) Findings: Café au lait spots (smooth “coast of California” border), Lisch nodules, optic pathway gliomas, Crowe sign

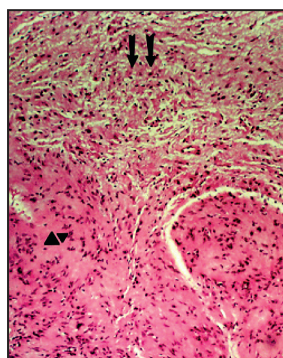


Fig 7-23 Schwannoma histology presenting Antoni A (triangles) and Antoni B (arrows) forms.

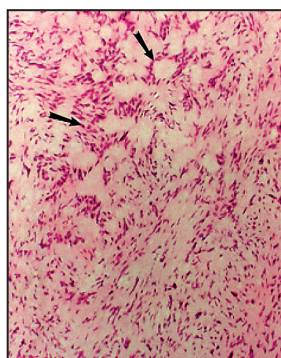


Fig 7-24 Schwannoma histology with Verocay bodies (arrows).

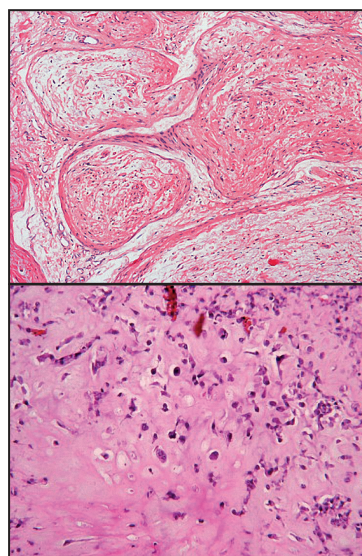


Fig 7-25 Plexiform neurofibroma.

Multiple endocrine neoplasia (MEN)

	Characteristics
Type 1: Wermer syndrome	Three Ps: Pituitary tumor, parathyroid tumor, pancreatic endocrine tumor
Type 2A: Sipple syndrome	Medullary thyroid carcinoma, pheochromocytoma, parathyroid tumor
Type 2B <ul style="list-style-type: none"> Autosomal dominant RET gene mutation 	Medullary thyroid carcinoma, pheochromocytoma, oral and/or intestinal neuromas

Melanotic neuroectodermal tumor of infancy

- Aggressive tumor of neural crest origin; osteolytic, pigmented neoplasm
- **Age:** Infant
- **Location:** Anterior maxilla
- **Clinical presentation:** Rapidly expanding, destructive bony lesion, which may appear blue on the surface
- **Radiograph:** Ill-defined radiolucency and tooth buds appear to be “floating”
- **Treatment:** Wide local excision
- **Prognosis:** 20% recurrence rate; malignant transformation is rare

Osteoid osteoma and osteoblastoma

Benign neoplastic proliferation of osteoblasts with low malignancy potential.

- Same pathology with different spectrum of disease severity; osteoblastoma is the less painful but more aggressive of the two lesions
- **Origin:** Gene alteration during osteoblast maturation (early in osteoblastoma; late in osteoid osteoma)
- **Histology:** Produces both osteoid and primitive woven bone amidst fibrovascular connective tissue stroma

	Osteoid osteoma	Osteoblastoma
Age	< 30 years	< 30 years
Clinical presentation	Painful local bony expansion	
Pain characteristics	Nocturnal pain (more intense)	Constant dull pain (less intense)
Pain relief with NSAID	Yes	No
Clinical aggressiveness (cellularity, growth, differentiation)	Less aggressive	More aggressive, less differentiated
Size	< 2 cm	> 2 cm
Recurrence	+	++
Treatment	Local excision	
NSAID, nonsteroidal anti-inflammatory drug.		

Osteoma

Benign neoplasm (hamartoma) that occurs almost exclusively in membranous bone; it is more common in the skull, not maxillofacial region.

- **Age:** Any age except children (unless it is associated with Gardner syndrome)
- **Histology:** Dense lamellar bone with little marrow
- **Clinical presentation**
 - Painless bony expansion (versus osteoblastoma)
 - Lesion is usually solitary with narrow base (versus torus or exostosis, multiple with broad base)
- **Treatment:** Local excision

Gardner syndrome

- Autosomal dominant: Mutation at chromosome 5
- Associated with
 - Multiple osteomas
 - Intestinal polyposis (often undergo malignant transformation)
 - Sebaceous/epidermal cysts of skin
 - Fibroma/fibromatosis of the soft tissues

Desmoplastic fibroma/aggressive fibromatosis

Rare benign myofibroblastic tumor with rapid growth and destructive behavior.

- Osseous form of aggressive fibromatosis
- **Age:** Average 15.1 years
- **Location:** Posterior mandible
- **Radiograph:** Not useful diagnostically; can be either well-defined or ill-defined radiolucency; cortical perforation; root resorption
- **Clinical presentation:** Asymptomatic or painful swelling, loose teeth, and/or trismus
- **Histology:** Benign appearance
 - Collagenous background with elongated and spindle-shaped fibroblasts
 - Indistinct cell borders and a cytoplasm that merges with the supporting collagenous background
- **Treatment:** “Treat biology, not histology;” resection with 1-cm margins
- Distinguish from low-grade fibrosarcoma

Chondroma

Benign tumor from cartilage cells.

- Extremely rare above clavicle; if present, one should question its diagnosis versus low-grade chondrosarcoma
- **Age:** > 20 years
- **Location**
 - Anterior maxilla from nasal septum
 - Mandibular condyle from articular fibrocartilage
 - Body of mandible from Meckel cartilage
- **Radiograph:** Well-defined radiolucency with foci of radiopacity
- **Clinical presentation:** Painless swelling
- **Treatment:** Resection with 1-cm margin
 - Concern for seeding in soft tissues
 - Rarity of this lesion in head and neck region may lead to confusion with low-grade chondrosarcoma

Malignant Nonodontogenic Tumors

Osteosarcoma

Malignant neoplasm arising from mesenchymal stem cells that exhibit osteoblastic differentiation and tumor bone production.

- Five histologic subtypes: No prognostic implications
 - Osteoblastic: Most common in the long bones
 - Chondroblastic: Most common in the jaws (Fig 7-26)
 - Fibroblastic
 - Telangiectatic
 - Small cell: Mimics Ewing sarcoma but forms osteoid (not in Ewing sarcoma)
- Two clinical subtypes
 - Central medullary osteosarcoma
 - Peripheral: Rare but better prognosis; does not penetrate to the medullary bone
 - Periosteal
 - Parosteal, more common
- **Age:** Average 27 years (older than osteosarcoma in long bones < 25 years)
- **Location:** Mandible > maxilla
- **Radiograph:** None of the findings are specific, and few osteosarcomas have all of these features
 - “Sun-ray” appearance (Fig 7-27)
 - Due to extracortical (right angle to the cortex) bone formation
 - In osteomyelitis bone formation parallel to the cortex
 - Widened periodontal ligament space: Garrington sign (Fig 7-28)



Fig 7-26 Chondroblastic osteosarcoma.

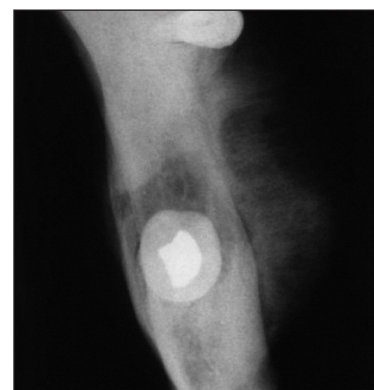


Fig 7-27 Osteosarcoma with sun-ray appearance.

- Codman triangle (Fig 7-29)
- Mottled radiopacity, mixed radiolucency, or opacity
- Cortical bone destruction and/or root resorption
- **Treatment** – Biopsy should be taken in the center of the lesion – No radiation (postradiation sarcoma) and no neck dissection; metastasizes via hematologic route – Multimodal treatment
 - Neoadjuvant chemotherapy not proven beneficial
 - Adjuvant chemotherapy typically administered
 - Resection with 2-cm margins (3-cm margins in long bones)
- **Prognosis:** 50% 5-year survival rate (versus 30% in long bones)



Fig 7-28 Garrington sign characteristic of an osteosarcoma.

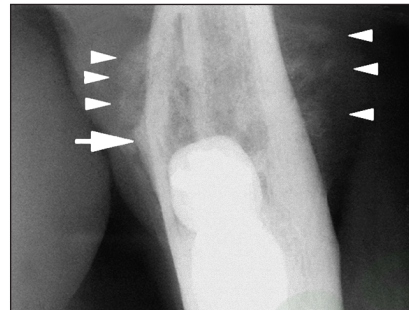


Fig 7-29 Codman triangle (*large arrow*) characteristic of an osteosarcoma.

Negative prognostic indicators

- Delay in diagnosis
- Increased tumor size
- Pain or paresthesia
- Tumor in the venules
- Myxomatous cells or tumor giant cells or necrosis
- Abundant calcified bone
- Positive margins in resection
- Nonmandibular location

Positive prognostic indicators

- Presence of cartilage cells
- Parosteal subtype

Ewing sarcoma

Malignancy of neuroectodermal origin; gene translocation t(11;22)(q24;q12).

- **Age/ethnicity:** Children and young adults; rare in blacks
- **Location:** Posterior mandible
- **Radiograph:** Ill-defined radiolucency; tooth displacement or root resorption
- **Histology:** Small round cell malignancy with prominent nuclei
- **Clinical presentation:** Extensive bony destruction and metastasis (lung and bone)
- **Treatment:** Multimodality therapy: Chemotherapy and/or surgery
- **Prognosis:** 5-year survival rate; 60% without metastasis; 30% with metastasis
- Positive prognostic indicators: Jaw > long bones; younger than 15 years old

Fibrosarcoma

Malignancy of connective tissue origin (periodontal ligament).

- **Age:** 2nd to 6th decade
- **Radiograph:** Ill-defined radiolucency; tooth displacement, resorption
- **Histology:** Malignant spindle cell tumor showing
 - Herring bone pattern (Fig 7-30)
 - Interlacing fascicular pattern
 - No expression of other connective tissue cells
- **Treatment:** Resection with 1- to 2-cm margins; benefits from chemotherapy or radiation therapy questionable
- **Survival rate:** 5 years; 71% (30% in long bones); minimal metastatic potential
- **Negative prognostic factors:** > 40 years; high histologic grade

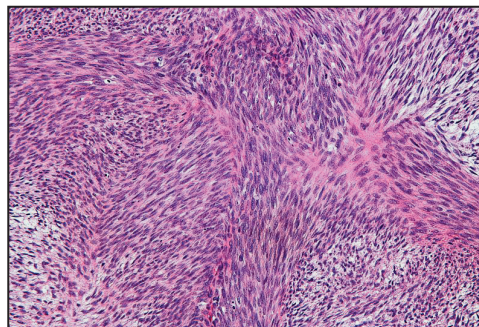


Fig 7-30 Herringbone pattern characteristic of a fibrosarcoma.

Chondrosarcoma

Malignant neoplasm arising from mesenchymal stem cells that partially differentiate into chondroblasts.

- **Age:** > 30 years
- **Location:** $\frac{2}{3}$ are osseous related; $\frac{1}{3}$ are soft tissue related
 - Anterior maxilla: From nasal cartilage
 - Posterior mandible: From Meckel cartilage
- **Clinical presentation:** Slow-growing mass; symptoms based on location
 - Nasal obstruction
 - Visual disturbance
 - Sensory alteration (neural invasion late in the course)
- **Radiograph:** Similar to osteosarcoma (sun-ray appearance, Garrington sign)
- **Histology**
 - Benign appearance or similar to chondroblastic osteosarcoma (cartilage formation but no osteoid or bone)
 - Grades 1 to 3 (based on mitotic rate, cellularity, and size of nuclei) correlates with prognosis
 - Grade 3: Increased tumor metastasis (lung)
 - Mesenchymal chondrosarcoma is a histologic subtype; presents in younger patients with faster growth and a tendency to spread (lungs)
- **Treatment:** Resection with 1- to 2-cm margins
- **Prognosis:** 50% 5-year survival rate
 - Long bone lesions have better prognosis than jaw lesions (except mesenchymal chondrosarcoma)
 - Prognosis is better than jaw osteosarcoma

Malignant peripheral nerve sheath tumor (MPNST)

- **Origin**
 - Preexisting neurofibroma (50% of MPNSTs develop from neurofibromatosis 1)
 - De novo
 - Postradiation sarcoma
- **Age:** Wide range from 4 to 76 years
- **Location:** Soft tissue; intraosseous (mandible)

- **Clinical presentation**
 - Paresthesia or anesthesia
 - Tooth mobility or bone expansion
- **Treatment:** Wide excision
- **Prognosis**
 - With neurofibromatosis: 5-year survival rate (16%)
 - Without neurofibromatosis: 5-year survival rate (53%)

Malignant fibrous histiocytoma (pleomorphic undifferentiated sarcoma)

Malignant neoplasm of soft tissue and bone.

- Can arise as primary tumor or as a secondary tumor from
 - Previously irradiated bone
 - Paget disease
 - Bone infarct
- **Age:** > 40 years
- **Clinical presentation:** Jaw expansion and/or tooth mobility
- **Radiograph:** Ill-defined radiolucency; tooth displacement, resorption
- Histologic subtypes (no prognostic significance)
 - Storiform pleomorphic spindle cell
 - Histiocytic
 - Multinucleated giant cell
- High-grade tumor likely to metastasize to the lung
- **Treatment:** Wide surgical resection
- **Prognosis:** Worse than its long bone counterpart

Burkitt lymphoma

High-grade, non-Hodgkin B cell lymphoma; c-myc oncogene activation via t(8:14).

- Two types
 - Endemic (African); related to human herpesvirus 4 (HHV-4)
 - Sporadic (American)
- **Age:** Endemic (3 to 8 years); sporadic (10 to 12 years)
- **Location:** Endemic (jaw is common; mandible > maxilla); sporadic (pelvis and/or abdomen)
- **Clinical presentation:** Tooth mobility, pain, or paresthesia
- **Radiograph:** Ill-defined radiolucency
- **Histology:** “Starry-sky” appearance; small B cells with multiple nucleoli (Fig 7-31)
- **Treatment:** Intensive, short-term, multi-agent chemotherapy
- **Prognosis:** 5-year survival rate 70% to 87%

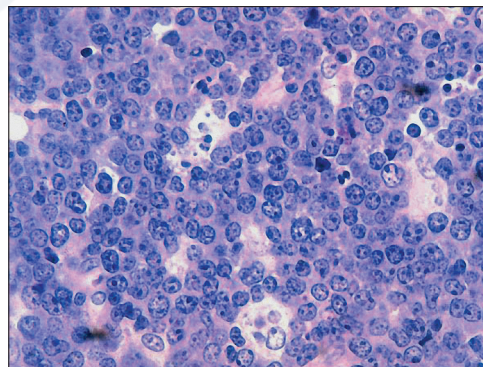


Fig 7-31 Starry-sky appearance of Burkitt lymphoma.

Multiple myeloma

- Malignancy of plasma cell origin
- **Age:** 60 to 70 years; blacks > whites
- **Histology:** Monotonous sheets of neoplastic, variably differentiated plasma cells
- **Radiograph:** “Punched-out” radiolucency (Fig 7-32)
- **Clinical presentation**
 - Jaw lesions only in 30% of patients
 - Bone pain
 - Pathologic fracture of involved bones
 - Hypercalcemia
 - Anemia
 - Petechiae: Platelet dysfunction
 - Amyloidosis in soft tissues
 - Renal failure from Bence-Jones protein (amyloid is nephrotoxic)
- **Treatment:** Chemotherapy and autogenous stem cell transplantation; intravenous bisphosphonates



Fig 7-32 Punched-out radiolucency of the calvarium in multiple myeloma.

Postradiation sarcoma

- More frequent in soft tissue than bone
- Dose dependent
- Average occurrence: 14 years after radiation
- Common preradiation pathology
 - Osteosarcoma
 - Malignant fibrous histiocytoma
 - Fibrosarcoma
- **Clinical presentation:** Variable but usually more aggressive and less responsive to treatment compared to the preradiation type of pathology
- **Treatment:** Wide surgical excision (2- to 3-cm margin)
- **Prognosis:** 30% survival for 5 years

Metastatic carcinoma of the jaws

- Primary tumor arises at distant sites: Breast > lung > renal > prostate > thyroid > colon > rectum
- Spreads via hematogenous route
- **Age:** 5th to 7th decades
- **Location:** Posterior mandible > maxilla
- **Clinical presentation:** Swelling, pain, paresthesia
- **Radiograph:** Irregular radiolucency except for metastases from breast and prostate (radiopacity)
- **Histology:** Depends on origin of the pathology; most of them are poorly differentiated
- **Treatment:** Poor prognosis; generally, palliative care with chemotherapy and radiation therapy

Odontogenic Cysts

Inflammatory	Developmental		Other
Periapical cyst Paradental cyst	Odontogenic <ul style="list-style-type: none"> • Primordial cyst • Dentigerous cyst • Gingival cyst of infants • Gingival cyst of adults • Glandular odontogenic cyst • Odontogenic keratocyst (OKC)* • Calcifying cystic odontogenic tumor† 	Nonodontogenic <ul style="list-style-type: none"> • Nasopalatine duct cyst (NPDC) • Median palatal fissure cyst 	<ul style="list-style-type: none"> • Simple idiopathic bone cavity • Mucous retention cyst

*The WHO has renamed OKC as keratocystic odontogenic tumor and classified it under odontogenic tumors.

†Calcifying odontogenic cyst (Gorlin cyst) has been renamed as calcifying cystic odontogenic tumor and is discussed under odontogenic tumors.

Inflammatory Cysts

Periapical cyst (radicular cyst, apical cyst, residual cyst)

- Most common odontogenic cyst
- Associated teeth are **not vital**
- Occurs from cystic degeneration of epithelial rests in a periapical granuloma
- Asymptomatic or pain, swelling, and drainage
- Well circumscribed radiolucency at the **apex of affected tooth**
- **Histology**
 - Cystic cavity with inflamed and reactive squamous cell epithelial lining
 - Cholesterol clefts
 - Multinucleated giant cells
- **Treatment**
 - Extraction or endodontic therapy
 - Enucleation and tooth extraction
 - Incomplete removal can lead to **residual cyst** formation and, rarely, intraosseous squamous cell carcinoma

Paradental cyst

- Associated with partially erupted third molars with pericoronitis
- Teeth are vital
- Well circumscribed radiolucency **adjacent to distal aspect of the crown of involved tooth**
- **Histology:** Similar to periapical cyst
- **Treatment:** Tooth extraction and curettage

Developmental Cysts (Odontogenic Origin)

	Origin	Clinical presentation and histology	Treatment	Comments
Primordial cyst	Enamel organ	<ul style="list-style-type: none"> Forms in place of normal or supernumerary tooth Radiolucency where a tooth did not form 	Enucleation and curettage	
Dentigerous cyst	Accumulation of fluid between crown and the reduced enamel epithelium	<ul style="list-style-type: none"> Third molar or canine region Tooth displacement, root resorption, bony expansion Radiograph: Well-defined pericoronal radiolucency 	<ul style="list-style-type: none"> Enucleation and curettage Removal of associated tooth Decompression before definitive treatment of large cysts 	<ul style="list-style-type: none"> Can give rise to ameloblastoma, squamous cell carcinoma, and mucoepidermoid carcinoma Soft tissue counterpart: Eruption cyst
Gingival cyst of infants	Remnants of dental lamina	<ul style="list-style-type: none"> Multiple smooth, white nodules along the alveolar crest Asymptomatic Histology: Keratin-filled cyst lined with stratified squamous epithelium 	Observation: Will rupture and involute	Differential diagnosis <ul style="list-style-type: none"> Epstein pearls: Mid-line palatal fissure cyst Bohn nodules: Blocked salivary duct located on hard palate often near junction with soft palate
Gingival cyst of adults	Rests of Serres (postfunctional dental lamina)	<ul style="list-style-type: none"> Bluish nodule on attached gingiva Canine or premolar region (mandible > maxilla) 	Excision	Soft tissue counterpart of lateral periodontal cyst
Lateral periodontal cyst (LPC)	Rests of Malassez	<ul style="list-style-type: none"> 66% in mandibular premolar or canine region Teeth are vital Histology: Glycogen-rich cells Radiograph: Lateral radiolucency in midroot region 	Enucleation and curettage	Botryoid odontogenic cyst (BOC), variant of LPC but more aggressive
Glandular odontogenic cyst	Glandular differentiation in odontogenic cyst	<ul style="list-style-type: none"> Anterior mandible Painless swelling Histology: Multicystic lesion partially lined by respiratory epithelial cells; mucous pooling (Fig 7-33) 	<ul style="list-style-type: none"> Wide local excision Conservative management, high recurrence rate 	Difficult to differentiate from low-grade mucoepidermoid carcinoma

(Developmental Cysts [Odontogenic Origin] cont)

	Origin	Clinical presentation and histology	Treatment	Comments
OKC*	<ul style="list-style-type: none"> Reduced dental lamina Mutations in the <i>PTCH</i> gene (Hedgehog signaling pathway) 	<ul style="list-style-type: none"> 2nd and 3rd decades of life Small cyst: Asymptomatic Large cyst: Painful swelling, cheeselike aspirate Most common in ramus region Radiograph <ul style="list-style-type: none"> 75% unilocular; 25% multilocular Tooth displacement, resorption, cortical thinning or perforation Histology <ul style="list-style-type: none"> Lined by thin para-keratinized stratified squamous epithelium (six to eight cell layers thick) Epithelium often separated from the basement membrane; rete ridges inconspicuous (Fig 7-34) 	<ul style="list-style-type: none"> Enucleation and curettage with peripheral ostectomy Decompression before definitive treatment for large cysts 	<ul style="list-style-type: none"> Multiple OKCs: Nevroid basal cell carcinoma syndrome (Gorlin-Goltz syndrome) Autosomal dominant (variable penetrance) Associated with <ul style="list-style-type: none"> Numerous basal cell carcinomas Palmar pits Ovarian fibromas Medulloblastoma Bifid ribs Calcified falx cerebri Hypertelorism Orthokeratinized odontogenic cyst <ul style="list-style-type: none"> Similar to OKC Less aggressive Lower recurrence rate

*Reclassified under odontogenic tumors due to genetic alteration seen in its pathogenesis.

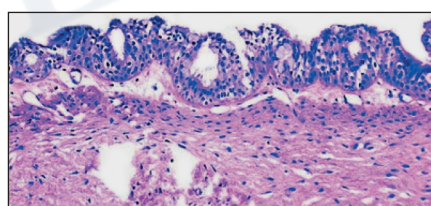


Fig 7-33 Glandular odontogenic cyst. Note the mucous cells within the epithelial lining.

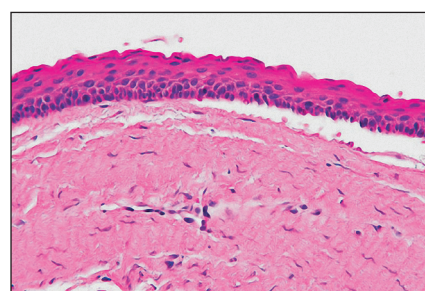


Fig 7-34 Odontogenic keratocyst showing thick cell layers and artifactual epithelial separation from basement membrane.

Developmental Cysts (Nonodontogenic Origin)

Nasopalatine duct cyst (NPDC)

Between 4th and 5th decades of life.

- Asymptomatic palatal swelling in anterior maxilla region; purulent drainage at times
- **Radiograph**
 - Well-demarcated round or pear-shaped radiolucency superimposed on incisive canal
 - Tooth displacement or divergence of the roots of the central incisors may be seen
 - Differentiate from incisive canal: > 6 mm in size in NPDC
- **Treatment:** Enucleation with low recurrence rate

Midline palatal cyst (Epstein pearls)

- Caused by epithelial entrapment during palatal development
- Seen in newborn
- Small white/yellow cystic vesicles in the median palatal raphe
- No treatment is needed; degenerate or rupture and disappear

Other Maxillofacial Cysts

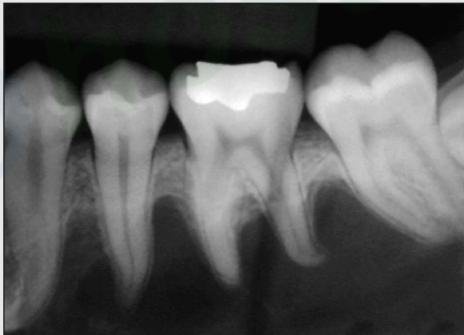
Traumatic bone cyst; idiopathic bone cavity	<ul style="list-style-type: none"> • Not a true cyst (no epithelial lining) • Always in mandible (posterior > anterior) • Young adults • Empty cavity or filled with a clear or serosanguinous fluid • Asymptomatic (70%); pain and lateral expansion (30%) • Teeth are vital • Radiograph <ul style="list-style-type: none"> – Well-demarcated radiolucency; scallops around the roots (Fig 7-35) – No tooth displacement and root resorption • Treatment: Observation or exploratory surgery to confirm empty cavity 	
Mucous retention cyst; antral pseudocyst	<ul style="list-style-type: none"> • Mucosal cyst of the maxillary antrum • Asymptomatic • Dome-shaped radiopacity on floor of the sinus • No tooth displacement and root resorption • Treatment: Observation 	

Fig 7-35 Traumatic bone cyst showing scalloping.

Non-neoplastic Salivary Gland Diseases

	Site	Pathology
Cystic lesion	Minor salivary gland	Mucocele
	Sublingual gland	<ul style="list-style-type: none"> • Ranula • Plunging ranula
	Parotid gland	<ul style="list-style-type: none"> • Lymphoepithelial cyst • Retention cyst • Hereditary polycystic disease • Parotid duct cyst
Reactive/inflammatory/infectious	<ul style="list-style-type: none"> • Bacterial/mycobacterial/parasitic infection • Viral infection • Autoimmune disease 	
Obstructive	Sialolithiasis	

Cystic Lesions

- These are not true cysts
- **Etiology:** Obstruction, congenital ductal abnormality, trauma
- Cysts in submandibular gland are rare

Site	Pathology	Pathogenesis	Clinical presentation	Treatment
Minor salivary gland	Mucocele	Traumatic severance of a minor salivary duct	<ul style="list-style-type: none"> • Lower lip: 63% of cases • Age: 1st through 3rd decades of life • Superficial saliva pooling; bluish vesicle • Deep saliva pooling; normal-colored nodule • Episodic enlargement and shrinkage 	<ul style="list-style-type: none"> • Local excision • Recurrence is common because of inadequate removal of the underlying involved salivary gland
Sublingual gland	Ranula	Injury or rupture of sublingual duct	<ul style="list-style-type: none"> • Translucent swelling in floor of mouth • Extension below mylohyoid is a plunging ranula (submandibular swelling) 	<ul style="list-style-type: none"> • Marsupialization (high recurrence rate) • Excision of sublingual gland
Parotid gland	Lymphoepithelial cyst	<ul style="list-style-type: none"> • Unknown • Associated with HIV and Sjögren syndrome 	Unilateral or bilateral painless parotid swelling	<ul style="list-style-type: none"> • Control HIV • Aspiration (high recurrence rate) • Superficial parotidectomy if cosmetically disfiguring

Salivary Gland Disorders: Inflammatory

Sialadenitis

- Inflammation of the salivary gland (parotid, submandibular)
- **Etiology** – Decreased salivary flow (ie, obstruction by a sialolith)
 - Decreased salivary production (ie, malnutrition, alcoholism, dehydration)
 - Congenital malformation of excretory duct – Infection (bacterial, viral, fungal, mycobacterial, and parasitic infections) or autoimmune (sarcoidosis, Sjögren syndrome)

Nonmodifiable factors	Relatively modifiable factors	Modifiable factors
Elderly age	<ul style="list-style-type: none"> • Anorexia nervosa/bulimia • HIV/AIDS • Cystic fibrosis • Endocrine disorder (diabetes, Cushing disease) • End-organ failure (kidney, liver) 	<ul style="list-style-type: none"> • Dehydration • Malnutrition • Medications • Recent surgery and anesthesia • Sialolithiasis

Clinical course

Timing and presentation	Age	Purulent
<ul style="list-style-type: none"> • Acute (< 1 month): Sudden swelling, overlying skin warm and red • Chronic (> 1 month): Gradual swelling • Recurrent 	<ul style="list-style-type: none"> • Juvenile • Adult 	<ul style="list-style-type: none"> • Suppurative (acute > chronic) • Nonsuppurative

Etiology

Infection	Autoimmune disease
<ul style="list-style-type: none"> • Acute allergic sialadenitis • Acute or chronic parotitis • Acute suppurative parotid or submandibular sialadenitis • Chronic recurrent juvenile parotitis or submandibular sialadenitis 	<ul style="list-style-type: none"> • Scleroderma • Dermatomyositis • Polymyositis • Systemic lupus erythematosus • Sjögren syndrome

(Etiology cont)

Infection	Autoimmune disease
Bacterial (community > hospital) <ul style="list-style-type: none"> Community-acquired: <i>Staphylococcus</i> or <i>Streptococcus</i> Hospital-acquired: <i>Staphylococcus</i> Treatment <ul style="list-style-type: none"> Hydration Digital massage Sialogogues Oral antibiotic (amoxicillin), outpatient Intravenous antibiotic (ampicillin), inpatient 	Sjögren Syndrome (SS) <ul style="list-style-type: none"> Middle-aged women Primary syndrome: Affects only the lacrimal and salivary glands Secondary syndrome: Primary syndrome with rheumatoid arthritis, possible systemic lupus erythematosus and/or scleroderma Increased risk of non-Hodgkin lymphoma Diagnosis based on the following <ul style="list-style-type: none"> Dryness of the eyes/mouth Schirmer test showing decreased tear production Labial gland biopsy (gold standard) Serology positive for SS, SS-A, and SS-B antigens Treatment – <ul style="list-style-type: none"> Supportive care Pilocarpine or cevimeline to increase saliva production Artificial tears and/or saliva Hydroxychloroquine for arthralgia and fatigue
Mycobacterial <ul style="list-style-type: none"> Tuberculosis (adult) Atypical mycobacteria (juvenile) <ul style="list-style-type: none"> <i>Mycobacterium kansasii</i> <i>Mycobacterium avium-intracellulare</i> 	
Viral <ul style="list-style-type: none"> Cytomegalovirus Mumps (paramyxovirus) <ul style="list-style-type: none"> Prodromal period 1 to 2 days Treatment: Supportive management; resolves in 5 to 10 days Complications <ul style="list-style-type: none"> Mumps pancreatitis Mumps orchitis (adult) HIV/AIDS – Diffuse infiltrative lymphocytosis syndrome (DILS) – All stages of HIV infection – Bilateral nontender swelling <ul style="list-style-type: none"> Treatment <ul style="list-style-type: none"> Antiretroviral drugs Sialogogues Hydration 	Sarcoidosis <ul style="list-style-type: none"> Chronic systemic noncaseating granulomatous disease of unknown origin Predilection for the lungs and hilar lymph nodes but can affect any body organ Salivary gland: Bilateral, firm, and painless parotid swelling; can develop before or concomitantly with other systemic symptoms Increased risk of non-Hodgkin lymphoma Heerfordt syndrome (uveoparotid fever) <ul style="list-style-type: none"> Manifestation of sarcoidosis – Consists of <ul style="list-style-type: none"> Uveitis Bilateral parotitis Facial nerve paralysis Treatment – Observation: Spontaneous symptom regression <ul style="list-style-type: none"> Steroid: Patients with decreased lung function or if the eyes, heart, kidneys, or spleen are severely involved Infliximab: Refractory sarcoidosis
Parasitic <ul style="list-style-type: none"> <i>Echinococcus granulosus</i> Suspect patient traveling from endemic areas (India) 	

Chronic recurrent juvenile parotitis

- Usually manifests **before puberty** and spontaneously resolves **prior to onset of puberty**
- Intermittent painful swelling of the parotid glands
- **Risk factors:** Congenital abnormalities of Stenson duct and history of mumps or trauma
- **Treatment:** Oral antibiotic (amoxicillin); conservative management

Obstructive Disease: Sialolithiasis

- **Etiology:** Stasis, infection, and changes in salivary constituents (pH, mucin content, ionic ratios)
- **Location:** Submandibular > parotid > minor > sublingual
- **Radiograph**
 - Submandibular: radiopaque stone (80% of the time)
 - Parotid: radiolucent stone (80% of the time)
- **Clinical presentation:** Asymptomatic (especially microlith) or intermittent swelling, pain, and purulent discharge at ductal orifice (from secondary ascending bacterial infection with *Staphylococcus aureus*)
- **Treatment** (based on the location of the stone and symptoms)
 - Asymptomatic patients with small stones: Observation – **Stone at distal end of the duct**, beyond mylohyoid for submandibular gland and masseter for parotid gland
 - Direct ductal incision or sialolithotomy with sialodochoplasty for submandibular stone; no duct closure for parotid stone
 - Fogarty catheter
 - Endoscopy
 - Lithotripsy
 - **Stone at proximal end of the duct**
 - Submandibular gland: Gland removal
 - Parotid: Superficial parotidectomy

Tumors of the Salivary Glands

- Arise from salivary acinar or ductal cells
- Rare with overall incidence of 2.5 to 3 per 100,000 per year
- **Location:** Parotid (80%), minor salivary gland (15%), submandibular gland (~5%), sublingual gland (~1%)
- Ratios of benign:malignant are site dependent
 - Parotid, 8:2 – Submandibular, 5:5 – Sublingual, 1:9 – Minor salivary gland
 - Soft and hard palate junction most common, 5:5
 - Tongue, 1:9
 - Upper lip, 8:2
- Most common benign tumor: Pleomorphic adenoma
- Most common malignant tumor: Mucoepidermoid carcinoma; adenoid cystic carcinoma (submandibular gland)
- Tumors with **perineural invasion:** Adenoid cystic carcinoma; polymorphous low-grade adenocarcinoma (PLGA), mucoepidermoid carcinoma

- Tumors with **perineural spread**: Adenoid cystic carcinoma; high-grade mucoepidermoid carcinoma
- 89% of parotid tumors are in the superficial lobe
- 44% of intraoral tumors are in the palate
- Most labial tumors are in the upper lip
- **Diagnosis**
 - Fine needle aspiration biopsy
 - Magnetic resonance imaging (MRI)
 - Computed tomography (CT) scan

Benign salivary gland tumors

Pleomorphic adenoma (mixed tumor)

- **Distribution**: Parotid > palate > submandibular gland
- **Age**: 3rd decade or older
- **Sex**: Females > males
- **Histology**: A combination of ductal cells, chondromyxoid matrix and myoepithelial cells (Fig 7-36)
- **Treatment**
 - Partial superficial parotidectomy with at least a 5-mm cuff of normal tissue versus superficial parotidectomy
 - Submandibular gland tumors treated with submandibular gland excision
 - Palatal tumors treated with periosteal sacrificing, wide local excision with 1-cm margins
- Recurrence rate : < 5%
- Malignancy rate: 6%

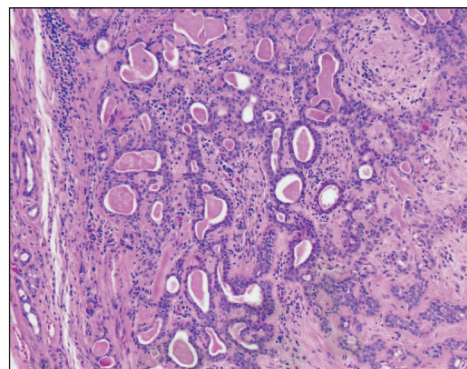


Fig 7-36 Pleomorphic adenoma showing tumor cells arranged in a ductal pattern. Note myxochondroid areas.

Papillary cystadenoma lymphomatosum (Warthin tumor)

- **Age**: > 40
- **Sex**: Males > females
- **Site**: Parotid; parotid tail most common
- 10% bilateral; 10% associated with smoking
- Painless and slow growing; frequently associated with other salivary gland tumors
- **Histology**: Papillary cystic growth of oncocytic ductal cells in a lymphoid stroma (Fig 7-37)
- **Treatment**: Partial superficial parotidectomy versus total superficial parotidectomy; low recurrence rate

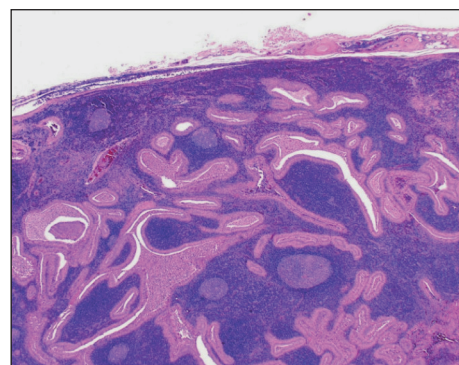
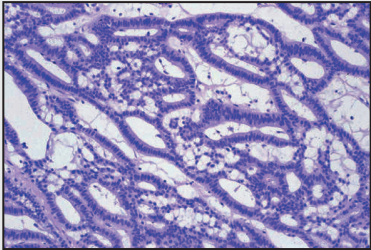
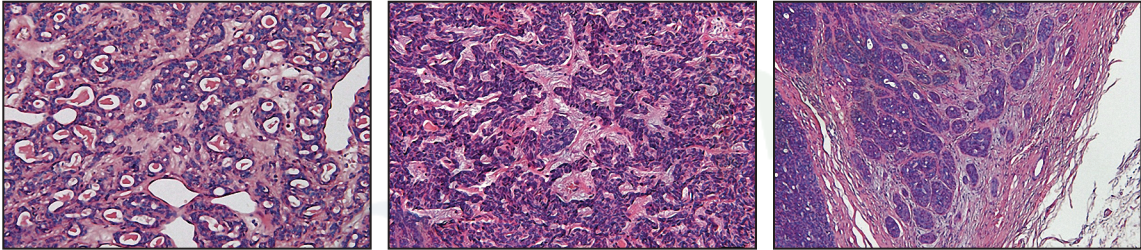
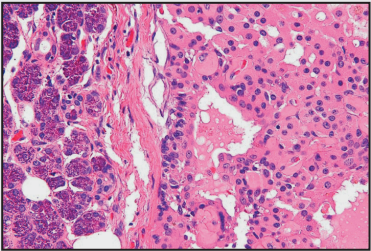


Fig 7-37 Warthin tumor showing lymphoid stroma containing cystic areas lined with oncocytic cells.

Monomorphic adenoma

- Asymptomatic swelling
- Multinodular presentation is not uncommon
- **Location**: 50% upper lip
- Most can be treated with local excision with minimal recurrence
- Subtypes: Canalicular, basal cell, oncocytoma, sebaceous, glycogen-rich, and clear cell adenoma

Canalicular adenoma	<ul style="list-style-type: none">• 75% are in the upper lip• 4th decade of life• Nonpainful submucosal nodule• Histology: Interconnecting strands of ductal cells that are often arranged in a double row (Fig 7-38)	
Basal cell adenoma	<ul style="list-style-type: none">• 75% in the parotid gland• 7th decade of life• Most common type• Basaloid-type cells with pale cytoplasm and indistinct cell boundaries• Encapsulated in major gland; absence of encapsulation in the minor glands• Many different histologic subtypes; no clinical significance (Fig 7-39)	
Oncocytoma	<ul style="list-style-type: none">• 80% in parotid gland• 7th decade of life• Nonpainful, firm, slow-growing mass• Histology (Fig 7-40)<ul style="list-style-type: none">– Similar to Warthin tumor but lack of stroma– Round eosinophilic polygonal cells with swollen cytoplasm and centralized nucleus• Treatment: Excision; low recurrence rate	

Other benign salivary gland tumors

Myoepithelioma	<ul style="list-style-type: none">• 5th decade• Histologically similar to mixed tumor but lacks myxoid pattern• Palate is the most common location
Ductal papilloma	<ul style="list-style-type: none">• Three histologic subtypes: Inverted duct, intraductal papilloma, sialadenoma papilliferum• Excision is the treatment of choice

Malignant salivary gland tumors

- Malignant salivary neoplasms present as a painless mass in approximately 75% of patients
- Palpable mass in a salivary gland associated with pain and/or nerve paralysis is more likely to be malignant than benign
- Episodic pain suggests obstruction, whereas constant pain is more suggestive of malignancy
- Trismus, cervical adenopathy, fixation of lesion, numbness, or bleeding also suggest the presence of malignancy
- **Prognosis**
 - High-stage or high-grade carcinomas have poor prognosis
 - Submandibular gland tumors have worse prognosis than parotid gland tumors
- **Treatment**
 - Total parotidectomy is the treatment for malignant tumors
 - Total submandibular or sublingual gland removal is indicated for both benign and malignant tumors in these locations
- Tumor, node, metastasis (TNM) classification
 - Similar to squamous cell carcinoma TNM system
 - T category can be subdivided into (a) no local extension and (b) local extension (eg, T3a)
 - Anything greater than T3a will be stage III; anything greater than T4a or N1 will be stage IV
- Indication for neck dissection: Prophylactic (level I to III only)
 - Positive clinical neck nodes: Modified radical neck dissection (level II to V) is warranted
 - High risk and/or grade pathology
 - T3 or T4 lesion
 - Age > 54
 - Perilymphatic invasion
 - Extraglandular spread

Low-grade malignant tumors	High-grade malignant tumors
<ul style="list-style-type: none"> • Low-grade mucoepidermoid carcinoma • Acinic cell adenocarcinoma • Adenoid cystic carcinoma • Polymorphous low-grade adenocarcinoma • Basal cell adenocarcinoma 	<ul style="list-style-type: none"> • High-grade mucoepidermoid carcinoma • Squamous cell carcinoma • Adenocarcinoma • Malignant mixed tumor • Malignant oncocyoma

Mucoepidermoid carcinoma (MEC)

- **Age:** 3rd decade
- **Clinical presentation**
 - Low-grade: Slow growing, painless mass
 - High-grade: Rapidly enlarging, possible pain
- Most common malignant salivary gland tumor
- **Location:** Parotid > palate > submandibular gland
- **Histology** (Figs 7-41 and 7-42)
 - Varying amounts of mucous, epidermoid, and glandular cells that may be arranged in solid/cystic pattern
 - High-grade MEC: **Epidermoid cells > mucous cells or glandular cells**

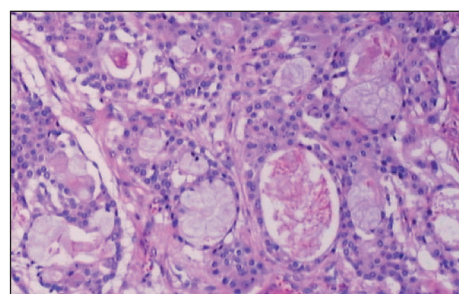


Fig 7-41 Low-grade mucoepidermoid carcinoma showing cystic spaces lined with epidermoid and mucous cells.

- **Treatment** – Stage I and II: Wide local excision or superficial parotidectomy
 - Stage III and IV
 - Wide excision or superficial parotidectomy
 - Possible neck dissection
 - Possible postoperative radiation therapy
- **Prognosis:** Histologic grading is important in determining prognosis
 - Low-grade: 85% to 100% 5-year cure rate
 - High-grade: 20% to 40% 5-year cure rate

Adenoid cystic carcinoma

- **Age:** 6th decade
- **Location:** Minor salivary gland (palate) > parotid gland > submandibular gland; most common malignancy in submandibular, sublingual, and minor salivary glands
- **Clinical presentation** – Asymptomatic enlarging mass – Slow growing, neurotropism, local recurrence, distant metastasis (early lung metastasis is common but may not influence overall survival)
 - Pain, paresthesia, facial weakness/paralysis
- **Histology** – Small uniform cells with dark, slightly angular nuclei – Three histologic subtypes
 - Cribriform: Most common (Fig 7-43)
 - Tubular: Ductlike structure (Fig 7-44)
 - Solid: Solid nests of cells without cystic or tubular spaces; worst prognosis (Fig 7-45)
- **Treatment:** Radical resection
- **Prognosis**
 - Indolent course due to perineural invasion
 - Local recurrence: 42%
 - 5-year survival rate is high; survival rate is 13% in 20 years

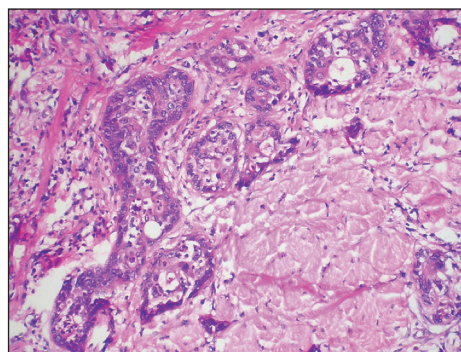


Fig 7-42 High-grade mucoepidermoid carcinoma showing atypical pleomorphic epithelial cells.

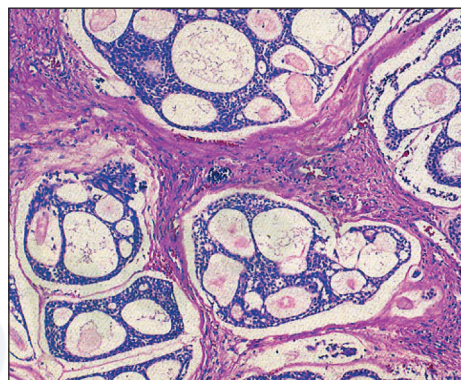


Fig 7-43 Adenoid cystic carcinoma showing the classic cribriform pattern.

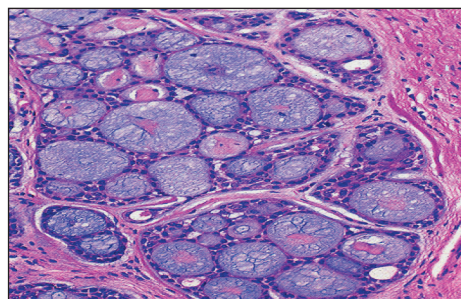


Fig 7-44 Tubular adenoid cystic carcinoma.

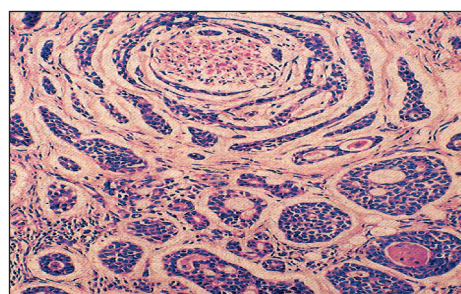


Fig 7-45 Adenoid cystic carcinoma showing perineural invasion.

Acinic cell adenocarcinoma

- **Age:** 3rd through 7th decade
- **Location:** Parotid gland > submandibular gland
- **Clinical presentation:** Swelling, pain, and tenderness are common
- **Histology** (Fig 7-46): Varying amounts of well-differentiated cells (acinous, intercalated duct, and glandular cells) that grow in solid, cystic, papillary, and follicular patterns
- **Treatment:** Wide excision is treatment of choice
- **Prognosis:** Good; 5-year survival rate > 80%

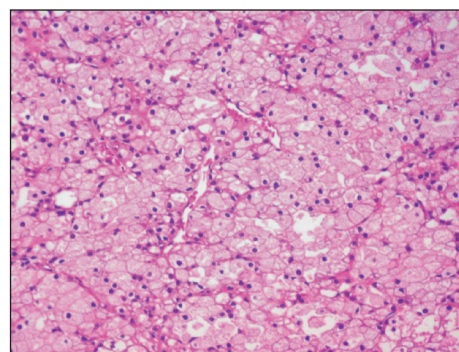


Fig 7-46 Acinic cell adenocarcinoma showing acinic and clear cells.

Other malignant salivary gland tumors

Polymorphous low-grade adenocarcinoma	<ul style="list-style-type: none"> • 6th decade • Asymptomatic swelling • Palate is the most common site • Frequently recur but rarely metastasize • Histology <ul style="list-style-type: none"> – Many growth patterns (polymorphous) within the same tumor: Solid, cystic, papillary, and single (Indian) file (Fig 7-47) – Perineural invasion • Wide excision; long-term follow-up 	A histological micrograph showing a pattern of cells arranged in a single file, which is characteristic of polymorphous low-grade adenocarcinoma. The cells are small and uniform, with minimal cytoplasm and dark nuclei.
Carcinoma ex pleomorphic adenoma	<ul style="list-style-type: none"> • Arises in mixed tumors of long duration (4.5% of mixed tumor) • Histology: Adenocarcinoma in a benign mixed tumor • 50% 5-year survival rate 	
Epithelial-myoepithelial carcinoma	<ul style="list-style-type: none"> • 7th decade • Rare lesion, occurs mostly in parotid gland • Histology: Polyhedral clear cells surround cuboidal ductal cells 	
Adenocarcinoma, not otherwise specified (NOS)	<ul style="list-style-type: none"> • Cytologic features and growth pattern do not allow classification • High-grade: 20% 5-year survival rate 	

Oral Squamous Cell Carcinoma

Epidemiology

- ~8,000 deaths/year
- ~37,000 new cases/year
- 66% of new cases are found in late stage (III or IV)

Risk factors

- Tobacco
- Alcohol
- Betel nut
- Human papillomavirus (HPV)

Precancerous lesions

- Leukoplakia: Malignant transformation rate ~ 6%
- Proliferative verrucous leukoplakia: Diffuse progressive leukoplakia; high frequency of progression to squamous cell carcinoma
- Erythroplakia: Malignant transformation rate ~ 25%
- Submucous fibrosis: Malignant transformation rate ~10%
- Erosive lichen planus: Malignant transformation rate ~<1%
- Hematopoietic stem cell transplantation: More aggressive behavior with poor prognosis

TNM Classification

- cTNM: Clinical staging based on findings before treatment
- pTNM: Histologic staging based on final specimen used to determine the need for adjuvant therapy

Tumor (T)

- Size: T1 < 2 cm; T2 between 2 and 4 cm; T3 > 4 cm; T4 bone erosion

Node (N)

- Size: N1 < 3; N2 between 3 and 6 cm; N3 > 6 cm
- Number: N2a for single node between 3 and 6 cm; N2b for multiple nodes all < 6 cm
- **Location:** N2c for contralateral or bilateral nodes < 6 cm

Metastasis (M)

- Stage 1: T1 N0 M0
- Stage 2: T2 N0 M0
- Stage 3: T3 +/- N1 or T1, T2N1 alone
- Stage 4: Any T4, any N2, any M1

Survival rate

- Stage I/II: > 80% 5-year survival rate
- One positive lymph node without extracapsular spread: 50% 5-year survival rate
- Positive extracapsular spread further decreases survival rate

Head and Neck Imaging

Typical oral squamous cell carcinoma (SCC) preoperative imaging includes

- Panoramic radiograph: Helps screen for bony invasion and nonrestorable teeth and used as follow-up screening tool for any osseous reconstruction
- CT/MRI face and neck with intravenous contrast: Preoperative tumor staging, evaluate osseous invasion, size and thickness of primary tumor
- Chest radiograph: Lung metastasis is the most common distant metastasis for oral SCC; this can be either conventional posteroanterior chest film or CT scan

Postoperative imaging should be done every 6 months in the first 2 to 3 years or whenever suspicious of recurrent disease.

Considerations

Tumor thickness	<ul style="list-style-type: none">• Important feature in deciding neck metastasis rate• MRI with contrast enhanced T1-weight fat-saturation image provides best estimate of tumor thickness in the tongue
Lymph node metastasis	<ul style="list-style-type: none">• CT and MRI have similar detection rate (approximately 80% to 90% sensitivity and specificity)• Metastatic lymph node characteristics<ul style="list-style-type: none">– Size: Level I and II should not be > 15 mm; Level III+ should not be > 10 mm– Central nodal necrosis– Extracapsular spread: Spiky, irregular margin, loss of fat cleavage plane around the node, thickening of adjacent fascia, and invasion of adjacent structures
Perineural spread	<ul style="list-style-type: none">• MRI provides 95% sensitivity detection rate• Characteristics<ul style="list-style-type: none">– Increase in nerve diameter– Destruction of the blood-nerve barrier and accumulation of contrast– Muscle atrophy provides other indirect evidence
Vascular invasion	<ul style="list-style-type: none">• CT image characteristics<ul style="list-style-type: none">– Tumor encasement of carotid circumference > 180 degrees– Partial effacement of fat plane or fascia between tumor and the artery• MRI image criteria for carotid invasion; carotid artery invasion is > 270 degrees
Mandibular invasion	<ul style="list-style-type: none">• CT has 96% sensitivity and 87% specificity• MRI with contrast 93% for both sensitivity and specificity (may overestimate the extent of the invasion due to inflammation and hemorrhage)
Previously treated neck	<ul style="list-style-type: none">• Distortion of the normal anatomy makes tumor surveillance difficult• Positron emission tomography (PET) scan has 88% success in detecting recurrent/residual cancer<ul style="list-style-type: none">– Wait at least 3 to 4 months after completion of radiation therapy, 3 to 7 days after biopsy, and 6 weeks after surgical resection for imaging

Neck Dissection Boundaries (Fig 7-48)

	Anterior	Posterior	Superior	Inferior
Level I	Anterior belly of digastric	Stylohyoid muscle	Mandible	Hyoid bone
Level II	Stylohyoid muscle	Posterior border of SCM	Skull base	Hyoid bone
Level III	Sternohyoid muscle	Posterior border of SCM	Hyoid bone	Inferior border of cricoid cartilage
Level IV	Sternohyoid muscle	Posterior border of SCM	Inferior border of cricoid cartilage	Clavicle
Level V	Posterior border of SCM	Anterior trapezius	Apex created by SCM and trapezius	Clavicle
Level VI	N/A	Carotid artery	Hyoid bone	Suprasternal notch

SCM, sternocleidomastoid.

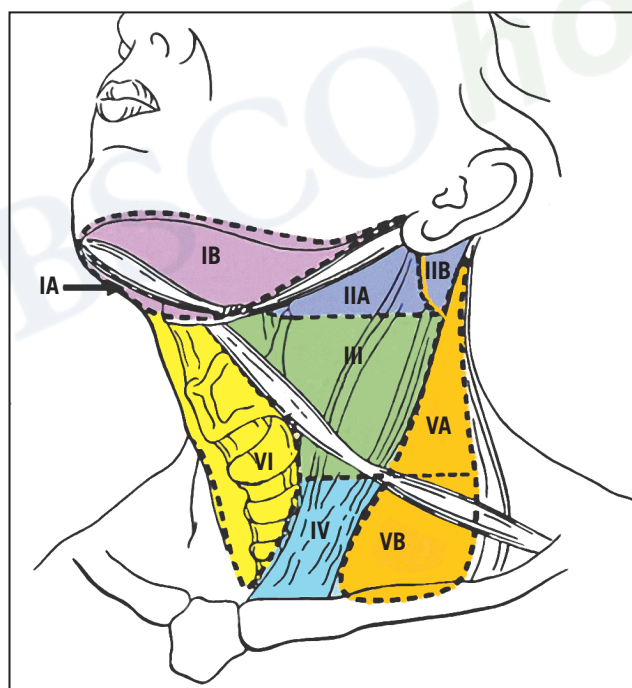


Fig 7-48 Neck dissection boundaries.

Neck Dissection

Indications

- Likelihood of occult metastasis to a regional lymph node is > 20%
- Tongue lesion with 3- to 4-mm depth
- Primary lesion is located in high-risk location: Floor of mouth, tongue, retromolar trigone
- T2 lesion or higher, some T1 cancers

Incision type

- MacFee incision
- Y incision

Complications

- Shoulder syndrome due to injury or removal of the spinal accessory nerve (SAN)
- Pain, weakness, deformity of shoulder girdle, inability to abduct the shoulder above 90 degrees
- Chyle leak
 - 1% to 2.5%, left side > right side due to presence of thoracic duct in the neck dissection field – Identify clear fluid intraoperatively by Valsalva maneuver and placing patient in Trendelenburg position
 - Identify postoperative: Milky drainage with triglycerides > 100 mg/dL
 - Management
 - < 500 cc/day: Conservative management with low fat diet (medium-chain triglycerides), elevation of the head, repeated aspiration and/or pressure dressings
 - > 500 cc/day: Medical management with octreotide

Dissection	Comment
Radical neck dissection (RND)	<ul style="list-style-type: none"> • Removal of all ipsilateral cervical lymph nodes • Levels I through V, together with SAN, sternocleidomastoid (SCM), and internal jugular vein (IJV)
Modified radical neck dissection (MRND)	<ul style="list-style-type: none"> • Similar to RND but with preservation of one or more nonlymphatic structures (SAN, SCM, and IJV) • Type I: Preserve SAN; most common for N+ oral cavity cancer • Type II: Preserve SAN and IJV • Type III: Preserve SAN, IJV, and SCM • Indicated when positive lymph node is found in the neck
Selective neck dissection (SND)	<ul style="list-style-type: none"> • Cervical lymphadenectomy with preservation of one or more lymph node groups that are routinely removed in an RND • Subtypes <ol style="list-style-type: none"> a. Supraomohyoid (level I to III); most common with oral cavity cancer b. Lateral c. Posterolateral d. Anterior • Oral cavity cancers: SND (I to III) • Oropharyngeal, hypopharyngeal, and laryngeal cancers: > SND (II to IV)

Neck metastasis rate*

Site	T1		T2		T3		T4	
	NO (%)	N+ (%)	NO (%)	N+ (%)	NO (%)	N+ (%)	NO (%)	N+ (%)
Tongue	86	14	70	30	52	48	23	77
Floor of mouth	89	11	71	29	56	44	46	54
Retromolar	88	12	62	38	46	54	32	78

*Data from Lindberg R. Distribution of cervical lymph node metastases from squamous cell carcinoma of the upper respiratory and digestive tracts. Cancer 1972;29:1446–1449.

Lymph node metastasis distribution (NO neck)*

- Most neck metastasis from oral SCC occurs in level I to III
- Skip node (isolated level IV) occurs < 3% to 15% of the time
- Positive lymph nodes in levels I to III will increase the positive rate of level IV nodes from 3% to 17%
- Oral SCC rarely metastasizes to level V; when level I to III nodes are positive, it only increases the likelihood of positive lymph nodes in level V from 0% to < 3%
- Level IIB is involved < 10% of the time in N0 neck patients

Site	Level I	Level II	Level III	Level IV	Level V
Tongue	27%	73%	18%	0%	0%
Floor of mouth	71%	43%	0%	0%	0%
Retromolar	25%	63%	12.5%	0%	0%
Mandibular gingiva	60%	40%	0%	0%	0%

*Data from Byers RM, Wolf PF, Ballantyne AJ. Rationale for elective modified neck dissection. Head Neck Surg 1998;10:160–167.

Adjuvant Treatment

- Based on clinical stage, patient health, and adverse features found preoperatively and postoperatively
- Adjuvant radiation therapy (RT) +/- chemotherapy is indicated when these adverse features are found
 - Extracapsular nodal spread and/or positive margins (requires concomitant chemoradiation therapy)
 - T3 or T4 primary
 - N2 or N3 nodal disease
 - Nodal disease in levels IV or V
 - Perineural invasion
 - Vascular embolism

T1 NO lesion

- Surgery or definitive RT; similar survival rate
- Residual disease after definitive RT; salvage surgery
- Adjuvant therapy indicated for surgical patients if adverse features are found

T2 T3 NO lesion

- Surgery with neck dissection
- Adjuvant therapy indicated for surgical patients if adverse features are found

T4a, any NO/N+ lesion

- Surgery and neck dissection
- N2c requires bilateral neck dissection
- Adjuvant therapy indicated for surgical patients if adverse features are found

Radiation therapy

- Dosage depends on
 - Primary disease and gross adenopathy: > 66 Gy
 - Uninvolved neck lymph node: ~60 Gy
 - Involved neck lymph node: ~ 63 Gy
- Administration methods –
Total dose is fractionated
 - To allow normal cells to recover
 - To allow remaining tumor cells to enter radiosensitive cell cycle
 - 1.8 to 2 Gy/day, 5 days a week, for the total of 7 weeks
- Modified technique
 - Hyperfractionation (concomitant boost): Two fractions per day of lower dose
 - Accelerated fractionation: Total dose of radiation is given over a shorter period due to two equivalent dose fractions per day
- Chemotherapy
 - Concurrent use with radiation therapy is better than radiation therapy alone

Chemotherapy

Neo-adjuvant therapy

- Role is limited in oral SCC, but more commonly used in laryngeal and hypopharyngeal cancer for organ preservation
- Commonly used in TPF regimen (docetaxel, cisplatin, and fluorouracil)
- No overall survival benefit

Primary therapy

- Used in nonresectable cases
- Target therapy and platinum-based therapy (EXTREME trial) in treating recurrent or metastatic head and neck SCC
 - Prolongs the medium overall survival rate by 7.4 months
- Target therapy and radiation therapy in advanced head and neck SCC
 - Prolongs local control by 10 months and overall survival 24 months versus radiation therapy alone

Adjuvant therapy together with radiation therapy (concurrent chemoradiation)

- Based on RTOG 95-01 and EORTC 22931
- Benefits only high-risk groups: Positive margin and extracapsular spread
- Improves both local control and disease specific survival
- Recommended regimen high-dose cisplatin (100 mg/m²) administered every 3 weeks
- 30% to 40% of patients cannot complete the course due to toxicity

Platinum based	<p>Mechanism of action</p> <ul style="list-style-type: none"> • Cell-cycle nonspecific alkylating agent • Binds to guanine on DNA, forming inter- and intrastrand crosslinks, inhibiting DNA synthesis <p>Toxicity</p> <ul style="list-style-type: none"> • Renal: Damage to the proximal and distal tubules; prevent by aggressive alkalinization of the urine and use of allopurinol • Neuropathic toxicity: Damage to the Schwann cell • Ototoxicity • Tumor lysis syndrome: Hyperkalemia, hyperphosphatemia, hyperuricemia, hypocalcemia, and metabolic acidosis <p>Most common platinum-based chemotherapy agents</p> <ul style="list-style-type: none"> • Cisplatin • Carboplatin <ul style="list-style-type: none"> – Related to cisplatin – 45 times less cytotoxic than cisplatin – More favorable adverse effect profile than cisplatin
Taxane	<p>Mechanism of action</p> <ul style="list-style-type: none"> • Binds to the B subunit of tubulin and stabilizes microtubules, which interrupts mitosis and leads to cell death <p>Toxicity</p> <ul style="list-style-type: none"> • Neutropenia: Usually dose limiting • Hypersensitivity: Dyspnea, urticaria, hypotension • Peripheral neuropathy, alopecia, bradycardia <p>Most common taxoid chemotherapy agents</p> <ul style="list-style-type: none"> • Docetaxel • Paclitaxel
Pyrimidine analog	<p>Mechanism of action</p> <ul style="list-style-type: none"> • Irreversible inhibition of thymidilate synthase leading to a lack of thymidine, imbalanced cell growth, and death <p>Toxicity</p> <ul style="list-style-type: none"> • Mucositis • Other side effects include bone marrow suppression, nausea and vomiting, alopecia, and anorexia <p>Most common used</p> <ul style="list-style-type: none"> • Fluorouracil

(Adjuvant therapy together with radiation therapy cont)

Target therapy	<p>Epidermal growth factor receptor (EGFR) inhibitor</p> <ul style="list-style-type: none"> • 80% to 90% of head and neck SCC show aberrant expression • High-volume expression correlates with poor prognosis <p>Cetuximab</p> <ul style="list-style-type: none"> • Mechanism of action <ul style="list-style-type: none"> – Recombinant monoclonal antibody – Binds to the extracellular domain of the EGF receptor to inhibit tyrosine kinase activation leading to apoptosis • Toxicity <ul style="list-style-type: none"> – Acneiform rash: Patients with prominent rash (grade 2 to 4) carry a better overall survival prognosis than patients with no or mild rash (grade 0 and 1) • Indications <ul style="list-style-type: none"> – Locally advanced head and neck SCC with radiation – Recurrent or metastatic disease with platinum-based chemotherapy
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Verrucous Carcinoma

- Noninvasive variant of squamous cell carcinoma of the oral cavity
- 3% to 4% of all oral carcinomas
- Sex: Females > males in oral cavity; males > females in larynx
- **Location:** Buccal mucosa and gingiva
- Precursors: Verrucous hyperplasia and proliferative verrucous leukoplakia

Clinical features

- Slow-growing, painless, broad-based verrucous or wart-like papillary lesion
- Locally invasive but no regional/distant metastasis

Histology

- Papillary invaginations of thickened and infolding epithelium (Fig 7-49)
- Minimal mitosis/atypia
- No violation of basement membrane

Management

- Wide excision
- Neck dissection and adjuvant therapy not needed

Prognosis

- > 80% 5-year survival rate
- 20% to 50% recurrence rate (close follow-up)

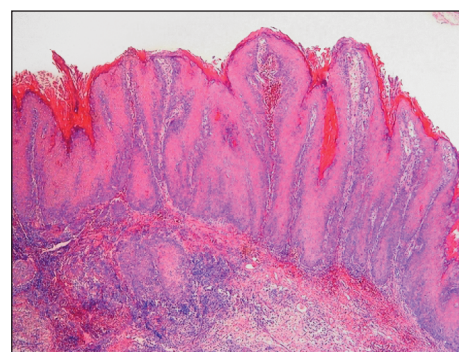


Fig 7-49 Histology of verrucous carcinoma showing characteristic papillary invagination and intact basement membrane.

Cutaneous Lesions

Lentigo

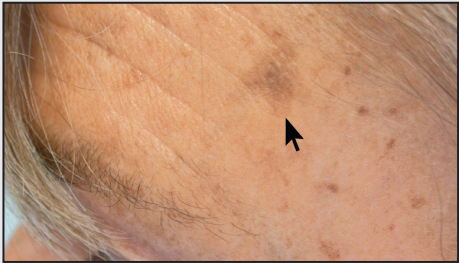
Solar lentigo	<ul style="list-style-type: none"> • Hyperpigmented lesion caused by increased melanin content, no change in number of melanocytes • Sun-exposed area • Tan-brown pigmentation • Multiple lesions up to 10 mm in size 	
Simple lentigo	<ul style="list-style-type: none"> • Localized proliferation of epidermal melanocytes • No association with sun-exposed area • < 5 mm in diameter • Evenly pigmented with darker color than solar lentigo 	

Fig 7-50 Solar lentigo.

Fig 7-51 Simple lentigo.

Nevus

Melanocytic nevus

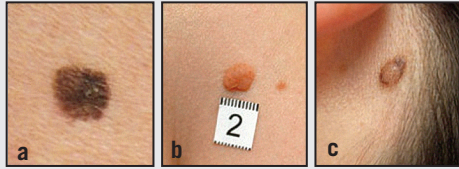
Congenital	Present at birth; only large lesions have potential for malignant transformation	
Acquired	<ul style="list-style-type: none"> • Three types <ul style="list-style-type: none"> – Junctional: Flat pigmented (above basement membrane) – Intradermal: Dome shaped (confined to dermis) – Compound: Slightly elevated • Uniform color, well-defined margin, and no history of bleeding/ulceration • No malignant potential 	

Fig 7-52 Acquired melanocytic nevus: junctional nevus (a), intradermal nevus (b), and compound nevus (c).

(Melanocytic nevus cont)

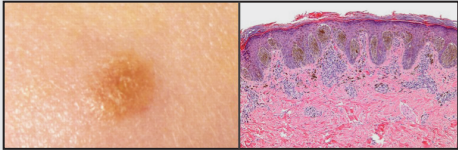


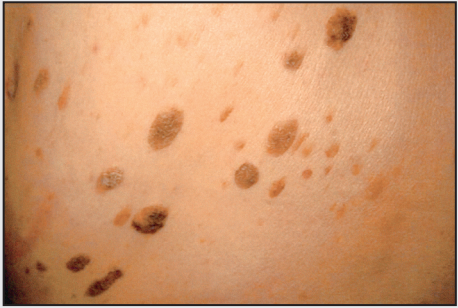
Spitz nevus	<ul style="list-style-type: none"> • Found mostly in children • Nevus cells that have both a spindled and epithelioid morphology (vertically arranged, “hanging bananas”) • No malignant potential 	
Halo nevus	<ul style="list-style-type: none"> • A central pigmentation is surrounded by a halo of depigmentation • Rare in head and neck, no malignant potential 	
Dermal melanocytoma	<ul style="list-style-type: none"> • Nevus cells confined within the dermis • Malignant potential reported but uncommon • Three major types <ul style="list-style-type: none"> – Blue nevus: Between 2- and 10-mm blue-black, symmetric, dome-shaped papule – Mongolian spot: Back or buttocks of infant – Nevus of Ota and Ito: Trigeminal distribution 	
Epidermal nevus	<ul style="list-style-type: none"> • Present at birth or develops in early childhood • Flat, tan patches of skin or raised, velvety patches • No malignant potential • Two major types <ol style="list-style-type: none"> 1. Nonorganoid epidermal nevus (contains keratinocytes only) 2. Organoid epidermal nevus (contains other epidermal cell structures such as hair follicles) 	
Seborrheic keratosis	<ul style="list-style-type: none"> • “Stuck-on” appearance • Usually < 1 cm in diameter • 4th decade and incidence increases with age • No malignant potential • Sudden onset of hundreds of seborrheic keratoses (Leser-Trelat sign) associated with internal malignancy 	

Fig 7-53 Splitz nevus.

Fig 7-54 Halo nevus.

Fig 7-55 Epidermal nevus.

Fig 7-56 Seborrheic keratosis.

Precancerous Cutaneous Lesions

	Actinic keratosis	Dysplastic melanocytic nevi
Histology	Atypical keratinocytes: Do not encompass the entire epidermis	<ul style="list-style-type: none"> Architectural atypia Cytologic (melanocytic) atypia
Malignant transformation	2% to 10% SCC	<ul style="list-style-type: none"> 30% of melanomas associated with a precursor nevus Single lesion: 2× risk for melanoma Presence of > 10 lesions (dysplastic nevus syndrome): 12× risk for melanoma
Clinical presentation	<ul style="list-style-type: none"> Sun-exposed area Rough erythematous papules with white/yellow scale (Fig 7-57) 	<ul style="list-style-type: none"> Trunk or upper back Larger than common nevus Macular and/or papular components Irregular, ill-defined borders (Fig 7-58) Variable tan to dark brown pigmentation
Treatment	<ul style="list-style-type: none"> Cryosurgery Topical chemotherapy 	Surgical excision with 0.5-mm margin



Fig 7-57 Actinic keratosis.

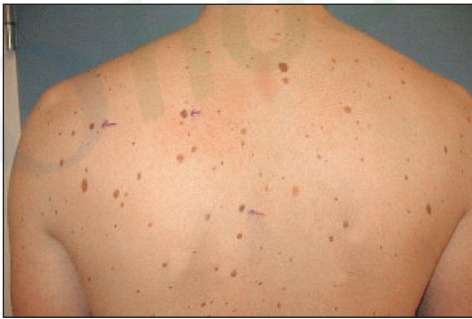


Fig 7-58 Dysplastic melanocytic nevus.

Nonmelanoma Skin Cancer

	Basal cell carcinoma (BCC)	SCC
Frequency	80% of all skin cancers	9% to 14% of all skin cancers
Age	Between 6th and 7th decades	
Etiology	<i>PTCH</i> gene mutation	p53 mutation

(Nonmelanoma Skin Cancer cont)

	Basal cell carcinoma (BCC)	SCC
Risk factors	<ul style="list-style-type: none"> • Fitzpatrick I to III skin type • Chronic sun exposure • Ultraviolet light exposure • Old age • Arsenic or tar exposure • Decreasing latitude • Immunosuppression (SCC > BCC) • Radiation • HPV 	
Subtypes	<ul style="list-style-type: none"> • Nodular (79%) • Superficial (15%) • Morpheaform (6%) • Micronodular {<1%} • Cystic • Basosquamous • Fibroepithelioma of Pinkus 	<ul style="list-style-type: none"> • In situ: Bowen disease (intraepidermal squamous cell carcinoma) • Keratoacanthoma
Clinical presentation	<ul style="list-style-type: none"> • Nodular (Fig 7-59) <ul style="list-style-type: none"> – Bleeds easily – Face > trunk • Superficial (Fig 7-60) <ul style="list-style-type: none"> – Erythematous scaly plaque – Trunk > face • Morpheaform (Fig 7-61): Resembles scar • Cystic <ul style="list-style-type: none"> – Clear or blue gray – Contains clear fluid • Basosquamous <ul style="list-style-type: none"> – Behaves like SCC – Aggressive – Increased metastasis rate 	<ul style="list-style-type: none"> • Erythematous nodule • Indurated lesion • Nonhealing ulceration (Fig 7-62)
Metastatic rate	<ul style="list-style-type: none"> • 1% • Highest during the 5 years after treatment • Recurrent risk <ul style="list-style-type: none"> – > 2 cm – Positive margin – Long duration – Central part of the face or ear – Aggressive histology 	<ul style="list-style-type: none"> • 5% • Increases with perineural involvement or immunosuppression • Recurrent risk (8% in 5 years) <ul style="list-style-type: none"> – > 2 cm – Positive margin – Long duration – Lip, eyelid, and ear – Aggressive histology

(Nonmelanoma Skin Cancer cont)

	Basal cell carcinoma (BCC)	SCC
Treatment	Mohs surgery with 95% cure rate; surgical resection with margin <ul style="list-style-type: none">< 1-cm lesion: 2- to 3-mm margin1- to 2-cm lesion: 3- to 5-mm margin> 2-cm lesion: 1-cm margin	
Prognosis	Good	Metastasis to lymph nodes: Survival rate decreases > 50%



Fig 7-59 Nodular BCCs.

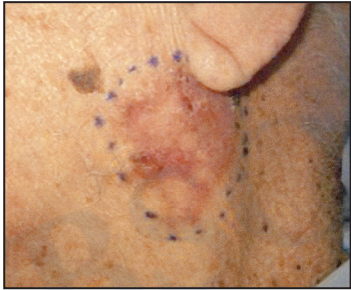
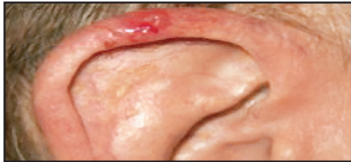


Fig 7-60 Superficial BCCs.



Fig 7-61 Morpheaform BCC.



Fig 7-62 Cutaneous SCC.

Melanoma

Frequency	4% of all skin malignancies
Etiology/pathophysiology	<ul style="list-style-type: none">Radial growth in the initial stageVertical growth in the late stage

(Melanoma cont)

Risk factors	<ul style="list-style-type: none"> • Fitzpatrick skin types I, II, and III • Intermittent sun exposure • Sunburns and tanning beds
Clinical presentation	<ul style="list-style-type: none"> • <u>A</u>symmetry • <u>B</u>order irregularity • <u>C</u>olor variegation • <u>D</u>iameter > 6 mm • <u>E</u>volution: a mole that has undergone recent change in color and/or size
Subtypes	<ul style="list-style-type: none"> • Superficial spreading melanoma—most common presentation; dark/pigmented macules (Fig 7-63) • Acral lentiginous melanoma (Fig 7-64) <ul style="list-style-type: none"> – Palm/soles; Hutchinson sign (pigmentation of proximal nail fold) – More common in dark-complexioned persons • Lentigo maligna melanoma (Fig 7-65) <ul style="list-style-type: none"> – Least common; but most common type on the face or sun-exposed area – Older patients • Nodular melanoma (Fig 7-66) <ul style="list-style-type: none"> – Second most common – – Raised blue-black-red papule or nodule – No radial growth stage, does not follow the “ABCD” rule

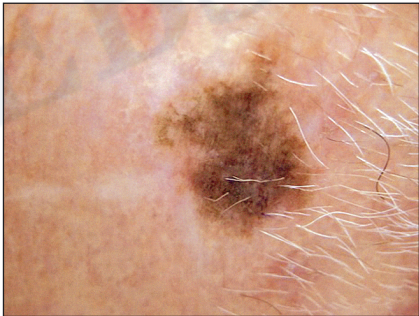


Fig 7-63 Superficial melanoma.



Fig 7-64 Acral lentiginous melanoma.

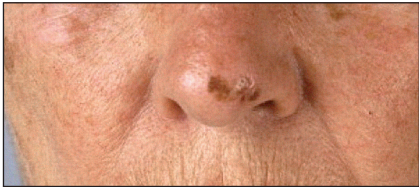


Fig 7-65 Lentigo maligna melanoma.



Fig 7-66 Nodular melanoma subtype.

(Melanoma cont)

Staging

- Breslow depth: based on depth in mm (Fig 7-67)
- Clark level: describes which layer of skin has been breached
- TNM staging (5-year survival rate)
 - Based on thickness, ulceration, anatomical site, and serum lactate dehydrogenase
 - Stage 0: In-situ; 99.9% survival rate
 - Stage I: Invasive melanoma; 89% to 95%
 - Stage II: High-risk melanoma; 45% to 79%
 - Stage III: Regional metastasis (lymph node, regional skin); 24% to 70%
 - Stage IV: Distant metastasis; 7% to 19%

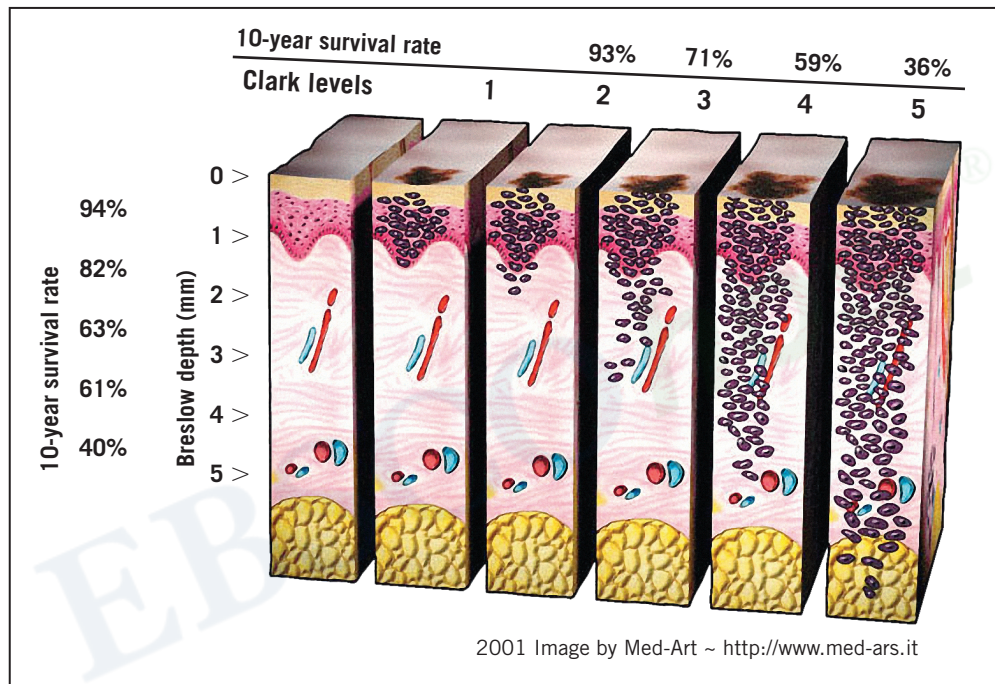


Fig 7-67 Breslow depth and Clark level staging. (Reprinted with permission of Med-Art.)

Treatment

- Surgical resection
 - In situ = 0.5- to 1-cm margin
 - 1-mm thickness = 1-cm margin
 - 1- to 4-mm thickness = 2-cm margin
- Sentinel lymph node biopsy
 - > 1-mm thickness
 - Ulceration
- Adjuvant therapy: Interferon alpha 2b

Other Skin Lesions


Comments	
Keratoacanthoma	<ul style="list-style-type: none">• SCC variant, low grade; occurs in sun-exposed areas• Rapid growth over weeks• Risk factors: Trauma, sun exposure, HPV 11 and 16• May progress to invasive SCC• Clinical presentation: Dome-shaped, red appearance with central hyperkeratotic core (Fig 7-68)• Histology: Similar to SCC• Treatment: May regress spontaneously; however, complete excision is warranted because of confusion with SCC clinically 
Merkel cell carcinoma	<ul style="list-style-type: none">• Older patient (> 75 years)• Aggressive skin tumor with early metastatic potential• Sun-exposed area• Associated with virus infection (Merkel cell polyomavirus)
Dermatofibrosarcoma protuberans	<ul style="list-style-type: none">• Cutaneous low-grade soft tissue sarcoma• 4th decade of life• Chromosomal translocation t(17;22)• Surgery or Mohs surgery is treatment of choice

Fig 7-68 Keratoacanthoma.

Mohs Surgery

- Microscopically controlled surgery
- **Four stages**
 1. Surgical removal of tissue
 2. Mapping/staining the specimen
 3. Histologic interpretation
 4. Further tissue removal/reconstruction
- **Indications**
 - Recurrent skin cancer (BCC or SCC)
 - Skin cancer in a high-risk anatomical area (ie, H zone on the face) (Fig 7-69)
 - Histologically aggressive skin cancer
 - Large skin cancer (> 3 cm) with ill-defined clinical margins
 - Incompletely excised skin cancer
 - Skin cancer in irradiated skin
 - Skin cancer in a cosmetically important area
 - Dermatofibrosarcoma protuberance (49% recurrence with conventional excision)



Fig 7-69 High-risk H zone on the face.

- **Contraindications**
 - Small lesions without danger to important cosmetic units
 - Malignant melanoma
 - High cost and time; each cycle takes ~ 45 minutes

Recurrence rates of BCC with different treatments

Treatment	Primary BCC	Recurrent BCC
Electrodesiccation and curettage	8%	40% to 59%
Cryosurgery	8%	8% to 19%
Radiation therapy	9%	9% to 51%
Surgical excision	10%	17%
Mohs surgery	1%	3% to 8%

Neck Masses

General Considerations

Many head and neck malignancies spread to the cervical lymph nodes. When an undiagnosed neck mass is encountered, it is always advisable to suspect a possible primary lesion elsewhere.

- < 40 years: 90% of lesions are benign
- > 40 years: “Rule of 80s,” 80% of nonthyroid neck masses are neoplastic; 80% of these masses are malignant
- Three categories: inflammatory, congenital mass, and neoplastic mass
- Differential diagnosis should be based on age and location
 - 0–15 years: Inflammatory > congenital > neoplastic (malignant) > benign
 - 16–40 years: Inflammatory > congenital > benign > malignant
 - 40+: Malignant > benign > inflammatory > congenital

Differential diagnosis

	Inflammatory mass	Congenital mass	Neoplastic mass
Age (years)	< 18	18–40	> 40
Duration	Acute (~ weeks)	Moderate	Chronic
Associated illness (URTI, pharyngitis)	+++	+	+/-
Constitutional symptoms	++	+/-	+
Associated pain	++	+	+
Functional symptoms (otalgia, dysphagia, facial nerve paralysis)	+	+/-	++
Social risk factors (smoking, alcohol)	-	+/-	+++
Immunocompromise	+++	+/-	+ (Kaposi sarcoma)

URTI, upper respiratory tract infection.

Differential diagnosis based on location

	Anterior triangle	Midline	Posterior triangle
Inflammatory	<ul style="list-style-type: none">• Sialadenitis<ul style="list-style-type: none">- Submandibular- Parotid	<ul style="list-style-type: none">• Plunging ranula• Thyroid mass	N/A
Infection	Lymphadenitis		
Congenital	<ul style="list-style-type: none">• Lymphangioma• Branchial cleft cyst	<ul style="list-style-type: none">• Thyroglossal duct cyst• Dermoid cyst	Lymphangioma
Neoplastic	<ul style="list-style-type: none">• Metastatic cancer• Lymphoma• Salivary gland tumor• Schwannoma• Carotid body tumor	<ul style="list-style-type: none">• Lymphoma• Thyroid mass	<ul style="list-style-type: none">• Lymphoma• Metastatic cancer

Infection

Bacterial

- Streptococcal and staphylococcal infection most common
- Cat-scratch disease
 - *Bartonella henselae*
 - 3 to 10 days after contact with infectious cat
 - Fever and tender, enlarged lymph nodes
 - **Treatment:** Cephalosporin and supportive management
- Mycobacterial
 - Atypical mycobacteria, seen more commonly in pediatric patient in the anterior triangle
 - Tuberculosis (scrofula)—suspect HIV—in the posterior triangle

Viral

- Epstein-Barr virus (EBV)
- Cytomegalovirus (CMV)
- Herpes simplex virus (HSV)
- HIV

Parasitic

- Toxoplasmosis from *Toxoplasma gondii*; found in uncooked meat or cat feces

Fungal

- Coccidiomycosis from *Coccidioides immitis*; endemic to southwestern United States; respiratory transmission

Congenital Lesions

Lymphangioma

- Present at birth but not noticeable until later in life
- Posterior triangle, behind SCM; (posterior to SCM—lymphangioma; anterior to SCM—brachial cleft cyst)
- Complete removal is not possible
- **Treatment:** Combination therapy (sclerotherapy and surgery) when lesion is disfiguring and/or impairs function

Branchial cleft cyst

- Epithelial cyst formation due to failure of obliteration of the second branchial cleft
- Anterior to SCM—branchial cleft cyst; posterior to SCM—lymphangioma
- Cyst wall is composed of either squamous or columnar cells with lymphoid infiltrate, often with prominent germinal centers
- Cholesterol crystals possible in the cystic fluid
- Most branchial cleft cysts are asymptomatic, but they may become infected (sudden increase in size)
- **Treatment:** Conservative management or surgery (recurrence may be high because complete resection is not always possible due to proximity to vital structures)
- Three types: first, second, and third branchial cleft cysts

	First branchial cleft cyst	Second branchial cleft cyst	Third branchial cleft cyst
Frequency	~4%–5%	95% (most common)	< 1%
Location	Originates at angle of the mandible and terminates in the external auditory canal	<ul style="list-style-type: none"> • Anterior to SCM • Inferior middle $\frac{2}{3}$ junction of SCM • Through carotid bifurcation and terminates in the tonsillar fossa 	<ul style="list-style-type: none"> • Anterior to SCM • Inferior middle $\frac{2}{3}$ junction of SCM (left > right) • Ascends behind the carotid sheath and terminates in the piriform sinus
Comment	Surgery may include superficial parotidectomy	Surgery may include tonsillectomy	Surgery may include thyroidectomy

Thyroglossal duct cyst

- Most common developmental cyst found in the neck
- Midline mass in the neck that moves during swallowing or tongue protrusion
- Swelling usually occurs after URTI
- Treatment: Removal of midportion of hyoid bone (Sistrunk procedure)

Dermoid cyst (cystic teratoma)

- Midline mass that does not elevate with tongue protrusion
- Painless, slow-growing lesion with doughy consistency
- Cystic structure with a lumen filled with keratin and a connective tissue wall with skin adnexa (all three germ cell layers)
- Treatment: Surgical excision

Thyroid Masses

Inflammatory

- Multinodular goiter
 - Physiologic, occurring during puberty or pregnancy
 - Secondary to living in endemic (iodine-poor) regions
 - Result of prolonged exposure to goitrogenic foods or drugs
- Hashimoto thyroiditis
 - Most common form of thyroiditis
 - Enlargement of the thyroid with possible pain and tenderness
 - Much more common in females (about 15% of US women)
 - Occasionally causes dysphagia or hypothyroidism
 - Serum titers of antimicrosomal and antithyroglobulin antibodies are elevated
 - Treatment: Small doses of thyroid hormone
 - Associated with lymphoma

Neoplastic

- 1.5% of all cancers in the United States
- Children: Malignant > benign
- Adult: Benign > malignant
- Leading cause of anterior neck masses
- Benign – Adenoma is the most common – Solitary, encapsulated, and compresses the adjacent thyroid gland – Removal is usually due to
 - Suspicion of cancer
 - Functional overactivity producing hyperthyroidism
 - Cosmetic disfigurement
- Malignant – History of irradiation to the neck – Family history of thyroid cancer – Firm or hard, fixed thyroid nodule; ipsilateral cervical lymphadenopathy – Types
 - Papillary adenocarcinoma: Most common; early adulthood; good prognosis; increased with childhood radiation; psammoma bodies
 - Follicular adenocarcinoma: Second most common; later in life; encapsulated; good prognosis
 - Medullary carcinoma
 - Genetically related; check family history
 - Isolated or associated with MEN type 2A or 2B
 - High incidence of nodal involvement
 - Secretes calcitonin
 - Anaplastic/undifferentiated
 - Poor prognosis
 - Older patients
 - High incidence of nodal involvement
 - Lymphoma
 - Associated with Hashimoto thyroiditis

Other Neoplastic Neck Lesions

Carotid body tumor (CBT)

- 65% of head and neck paragangliomas (neuroendocrine neoplasm)
- Develops within the adventitia of the medial aspect of the carotid bifurcation
- Asymptomatic palpable neck mass in the anterior triangle of the neck
- Fontaine sign: Mass is typically vertically fixed because of its attachment to the bifurcation of the common carotid; movable in anteroposterior direction
- Symptoms may be due to compression of surrounding nerves: Tongue paresis, hoarseness, Horner syndrome, dysphagia
- **Treatment**
 - Resection; embolization before surgery is needed for large tumors
 - Radiation therapy is an alternative for nonsurgical candidates

Vesiculobullous Diseases

These are diseases that present on the mucous membranes and/or skin as fluid-filled vesicles or bullae. The difference between bullae and vesicles is simply their size. The term *bulla* is applied to a lesion greater than 5 mm in diameter, while a *vesicle* is 5 mm or smaller in size. There are three major etiologies: immune related, hereditary, and infection.

Immune Related

	Pemphigus	Pemphigoid	Oral lichen planus
Age (decade)	4th to 6th	6th to 8th	5th and 6th
Sex	Equal	Equal	Female > male
Histology	Intraepithelial clefting (Fig 7-70)	Subepithelial clefting (Fig 7-71)	<ul style="list-style-type: none">• Hyperkeratosis• Saw-toothed rete ridges• Infiltrate of lymphocytes in the subepithelial layer (Fig 7-72)

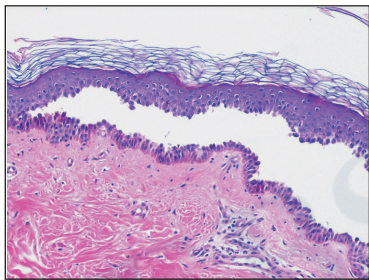


Fig 7-70 Intraepithelial clefting characteristic of pemphigus.

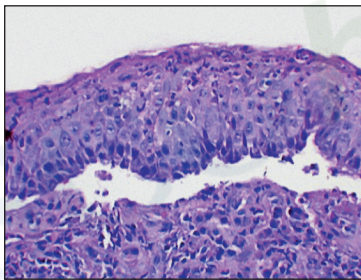


Fig 7-71 Subepithelial clefting characteristic of pemphigoid.

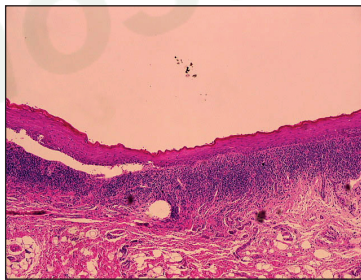


Fig 7-72 Oral lichen planus histology shows lymphocyte infiltration in the subepithelial layer.

Direct immunofluorescence	(+) Intracellular	(+) Basement membrane	(+) Fibrinogen
Subtypes	<ul style="list-style-type: none">• Pemphigus vulgaris• Paraneoplastic pemphigus	<ul style="list-style-type: none">• Bullous pemphigoid (BP)• Cicatricial pemphigoid (CP)	<ul style="list-style-type: none">• Reticular• Erosive• Bullous
Nikolsky sign	+	– BP or + CP	+
Location	Mucosa and/or skin	BP—skin CP—mucosa	Mucosa and/or skin

(Immune Related cont)

	Pemphigus	Pemphigoid	Oral lichen planus
Clinical features	<ul style="list-style-type: none"> • Painful ulcers or bullae • Bullae are rapidly ruptured, leaving a collapsed roof of grayish membrane with a red ulcerated base • Ulcers can become confluent • Can be life threatening 	<ul style="list-style-type: none"> • Oral mucosa is the first site; lesions are rarely widespread • Bleeding in the bullae; bleeding blisters • Ocular involvement; blindness 	<ul style="list-style-type: none"> • Reticular – Most common <ul style="list-style-type: none"> – Asymptomatic – Wickham striae (Fig 7-73) – Can occur anywhere in the mouth • Bullous – Fluid-filled vesicles <ul style="list-style-type: none"> – Similar to pemphigus and pemphigoid • Erosive <ul style="list-style-type: none"> – Erythematous areas that are ulcerated – Located mostly on buccal mucosa and/or gingiva – Resembles desquamative gingivitis – Only subtype that has ~1% malignancy transformation rate
Treatment	Supportive management	Topical corticosteroids (triamcinolone); for ocular involvement—systemic steroids	<ul style="list-style-type: none"> • First line: topical triamcinolone • Second line <ul style="list-style-type: none"> – Oral retinoids – Tacrolimus

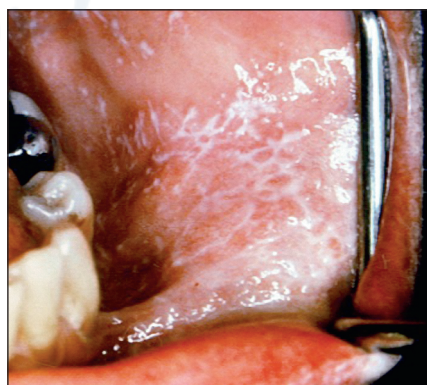


Fig 7-73 Wickham striae indicative of oral lichen planus.

Diseases with positive Nikolsky sign

- Pemphigus
- Cicatricial pemphigoid
- Oral lichen planus
- Epidermolysis bullosa
- Systemic lupus erythematosus
- Linear immunoglobulin A (IgA) dermatosis
- Graft versus host disease
- Toxic epidermal necrolysis

Erythema multiforme

- IgM-mediated immune disorder (type III hypersensitivity)
- Stimulated by
 - Infection (HSV, mycoplasma)
 - Drug exposure (sulfonamides, anticonvulsant)
- **Clinical presentation**
 - Epidermis detaches from the underlying tissue
 - Blister/erosion in mucosal tissue (mouth, lip, conjunctiva, genital, and anal regions)
 - Distinctive targetoid skin rash (Fig 7-74)
- Condition varies based on the level of epidermal detachment
 - Mild: Erythema multiforme (Fig 7-75)
 - Moderate: Stevens-Johnson syndrome (SJS)
 - Severe: Toxic epidermal necrolysis (TEN)
- **Treatment**
 - Discontinue all offending medications
 - Macrolides and/or doxycycline for mycoplasma infection
 - Supportive management: Intravenous fluid, nasogastric tube feeding, and pain management
 - Controversial: Systemic steroid and intravenous immunoglobulin (IVIG) therapy



Fig 7-74 Erythema multiforme with distinctive targetoid skin rash.



Fig 7-75 Oral presentation of erythema multiforme.

Hereditary

Epidermolysis bullosa (EB)

- Inherited connective tissue disease causing blisters in the skin and mucous membranes
- Incidence of 1/50,000
- **Etiology:** Genetic defects that lead to deficiency in the linking protein that anchors the epidermis to the underlying dermis
- **Clinical presentation**
 - Formation of blisters following trivial trauma
 - Healing leads to scar tissue formation; limited joint movement and/or mouth opening
- Three hereditary subtypes: EB simplex, junction EB, and dystrophic EB

	Genetic pattern	Separation level	Defective structure	Comment
EB simplex	A. Dominant	Intraepithelial	Linking protein	<ul style="list-style-type: none">• Most common• Mild form• Teeth are not involved
Junction EB	A. Recessive	Lamina lucida	Anchoring filament	<ul style="list-style-type: none">• Severe form• Teeth; hypoplasia
Dystrophic EB	A. Dominant A. Recessive	Sublamina densa	Type VII collagen	<ul style="list-style-type: none">• A. Recessive is severe• Teeth<ul style="list-style-type: none">– Delayed eruption– Enamel hypoplasia

Treatment

- No specific treatment available for hereditary types
- Maintenance of patient's nutritional and oral hygiene status
- Wound healing care
- Prevention of infections

Infection

	Disease	Clinical presentation
HSV	Herpetic gingivostomatitis	Herpetic gingivostomatitis often the initial presentation during the first herpes infection
	Herpes labialis (cold sore)	<ul style="list-style-type: none">• Can be primary presentation or viral reactivation• Self limited• Fever can occur• Prodromal symptoms; blister; ulcer• Acyclovir; given if diagnosed before blister formation• Supportive care if diagnosed after blister formation

(Infection cont)

	Disease	Clinical presentation
Herpes zoster virus (HZV)	Chicken pox	Vesicular rash begins on trunk; spreads to face and extremities, with lesions of different age
	Shingles	<ul style="list-style-type: none"> • Reactivation of HZV • Painful skin rash with blisters in a limited area (does not cross midline)
Coxsackie virus	Herpangina (Fig 7-76a)	Ulcers in oral cavity, usually confined to the posterior pharynx (tonsil and soft palate)
	Hand-foot-mouth (Fig 7-76b)	<ul style="list-style-type: none"> • Vesicular rash on palms and soles • Ulcers in oral mucosa (not confined to posterior pharynx)

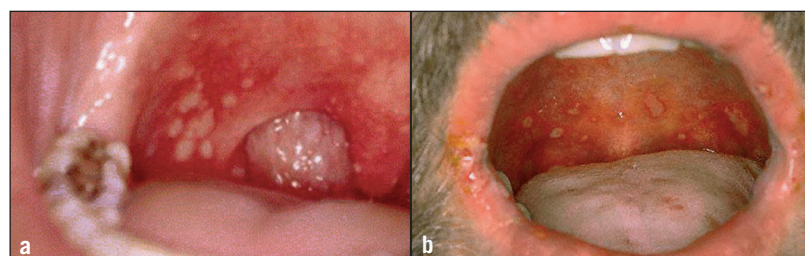


Fig 7-76 Clinical presentation of herpangina (a) and hand-foot-mouth disease (b).

Aphthous stomatitis (canker sore)

- T-cell-mediated immune response
- Trigger factors: Stress, local trauma (dental procedure), hormonal imbalance, nutritional deficiency
- Three types – Minor aphthous ulcerations
 - Most common; least severe
 - Occurs in childhood and adolescence
 - Exclusively on nonkeratinized mucosa (floor of mouth, buccal mucosa, soft palate)
 - Ulcer usually yellow-gray in color, with erythematous halo, less than 10 mm in size
- Major aphthous ulcerations
 - Typically 10 mm or larger in size
 - Painful and typically leave a scar
 - Take up to 1 month to heal
- Herpetiform aphthous ulcerations
 - Most severe form
 - Occurs frequently in females
 - Onset usually in adulthood
 - Small numerous lesions of 1–3 mm, often in clusters
- Supportive management
- Recurrence of multiple ulcers may require ruling out systemic disease
 - Behcet syndrome
 - Inflammatory bowel disease
 - Reiter disease

Vascular Anomalies

	Hemangioma	Vascular malformation
Onset	<ul style="list-style-type: none"> • 1/3 present at birth • 2/3 present postnatally 	Mostly at birth
Growth characteristic	Rapid growth in early infancy	Expands commensurately with growth of the child
Histology	<ul style="list-style-type: none"> • Endothelial hyperplasia • GLUT-1 positive • Increased mast cell count 	<ul style="list-style-type: none"> • Slow endothelial turnover • GLUT-1 negative • Normal mast cell count
Subtype	<ul style="list-style-type: none"> • Infantile hemangioma • Congenital hemangioma <ul style="list-style-type: none"> – Rapidly involuting (RICH) – Noninvoluting (NICH) 	<ul style="list-style-type: none"> • Low flow <ul style="list-style-type: none"> – Capillary (CM) – Lymphatic (LM) – Venous (VM) • High flow <ul style="list-style-type: none"> – Arteriovenous malformation (AVM)
Skeletal deformity	None	Low flow: Bony deformity High flow: Osteolysis
Hematologic findings	None	Vascular stasis Localized/systemic DIC
Syndrome	<ul style="list-style-type: none"> • PHACE • Kasabach-Merritt syndrome 	Sturge-Weber syndrome
GLUT-1, glucose transporter 1; DIC, disseminated intravascular coagulation; PHACE, posterior fossa malformation–hemangioma–arterial cerebrovascular anomalies–coarctation of the aorta–eye/endocrine disorder.		

Infantile Hemangioma

- Presents postnatally
- Most common benign tumor of childhood (7% of all soft tissue tumors)
- Present in 8% to 12% of children
- Female to male ratio of 3:1
- Risk factors
 - Premature birth
 - < 1,200 kg birth weight
- Stages
 - Proliferation (6–9 months)
 - Involution (10% per year)
 - Involuting (50% by age of 5 years; 70% by age of 7 years)

Clinical presentation

Rapid growth with slow regression.

- Proliferation phase (6–9 months)
 - Firm to palpation, but no pulsation or thrill
 - Superficial, erythematous, macular patch
 - Deep bluish hue with normal overlying skin
- Involution phase (12 months to 10 years)
 - Color fades
 - Shrinks and flattens
- Involved phase (5 years +)
 - 50% of children have residual abnormalities
 - Redundant atrophic skin
 - Yellow discoloration
 - Residual fibrofatty tissue
 - Telangiectasia

PHACE syndrome

- P: Posterior fossa brain malformation
- H: Hemangioma
- A: Arterial cerebrovascular anomalies
- C: Coarctation of the aorta
- E: Eye/Endocrine disorder
- 8% of PHACE children will have a stroke in infancy

Management

- Observation: Monthly during the proliferation stage; annually during involution stage
- Intervention indicated when lesion causes destruction, distortion, or obstruction –
 - Intralesional steroid (3 mg/kg triamcinolone), oral propranolol, or intralesional bleomycin
 - Reserve for lesion < 2 cm in size
 - Lasts for 4–6 weeks
 - Systemic steroid or intralesional vincristine
 - Reserve for large destructive lesions not impinging on vital structures
 - Surgery
 - Proliferation stage: When vital structure is involved
 - Involved stage: Revision surgery

Congenital Hemangioma

- Vascular tumors that are fully grown at birth
- Do not exhibit same life cycle as infantile hemangioma
- Similar management as infantile hemangioma
- Two types
 - RICH
 - NICH

	RICH	NICH
Life cycle	Involutes within the first few weeks/months of life	None
Color	Red	Gray
Characteristic	Ulceration with pale halo	Well circumscribed
Location	Trunk/extremities	Mandibular border

Vascular Malformation

- Present at birth and expands commensurate with child growth
- Usually not noticeable until later in life
- 31% of these malformations are found in the head and neck region
- Trauma, infection, and hormonal fluctuation (pregnancy or puberty) may stimulate increased growth of the vascular malformation
- MRI is the gold standard to determine the type and extent of the lesion
- Two types
 - Low flow
 - More common type
 - CM
 - LM
 - VM
 - High flow
 - AVM

Management

- Multidisciplinary approach
- Small lesion: Laser therapy/surgical resection alone
- Large lesion: Combination therapy
 - Sclerotherapy (low-flow lesion)
 - Embolization (high-flow lesion)
 - Surgery: Debulking
- Malocclusion/asymmetry: Orthognathic surgery
- Airway compromise (cervicofacial LM): Tracheotomy

Prognosis

- Recurrence is high
- Complete eradication is not possible due to proximity to vital structures

	Low flow			High flow
	CM	LM	VM	AVM
Synonyms	Port-wine stain	Cystic hygroma (LM in the neck)	N/A	N/A

(Prognosis cont)

	Low flow			High flow
	CM	LM	VM	AVM
Presentation	<ul style="list-style-type: none"> • Dermatome distribution • 45% in one of the trigeminal nerve distributions • Increased hue with age: red (young adult) and purple (middle age) • Common to have labial/gingival hypertrophy and maxillary cant 	Types <ul style="list-style-type: none"> • Macrocytic (not compressible) • Microcytic • Mixed • Cervicofacial LM <ul style="list-style-type: none"> – Soft tissue hypertrophy – Bony enlargement (mandibular overgrowth) 	<ul style="list-style-type: none"> • Most common type • Thin walled • Dilated channel • Normal endothelial lining • Deficient smooth muscle • Causes malocclusion • Phleboliths and thromboemboli are uncommon 	<ul style="list-style-type: none"> • Abnormal communication between arteries and veins (without capillaries) • Schobinger staging <ul style="list-style-type: none"> – Stage I: Quiescent— asymptomatic – Stage II: Expansion— asymptomatic expansion – Stage III: Destruction— expansion + pain + bleeding + tissue necrosis – Stage IV: Decom-
Prognosis	<ul style="list-style-type: none"> • Difficult to eradicate • May require life-long treatment 	<ul style="list-style-type: none"> • Difficult to eradicate • Recurrence is high 	<ul style="list-style-type: none"> • Recurrence is high • Difficult to eradicate due to close proximity to vital structures 	<ul style="list-style-type: none"> • Complete resection often not possible • Recurrence in 1 year and re-expansion within 5 years
Management	<ul style="list-style-type: none"> • Intervention for cosmetic/functional purposes • Skin: Multiple laser therapies • Malocclusion: Orthognathic surgery • Labial/gingival hypertrophy: Contour resection (no concern for excessive bleeding) 	<ul style="list-style-type: none"> • Small and/or asymptomatic <ul style="list-style-type: none"> – Pain medication – Rest – Antibiotic (daily if > 3 infections per year) – Surgery if lesion is small • Large and/or symptomatic <ul style="list-style-type: none"> – Combination therapy with <ul style="list-style-type: none"> ◦ Sclerotherapy (ethanol, bleomycin, and doxycycline) ◦ Surgery ◦ Tracheotomy, if airway compromised 	<ul style="list-style-type: none"> • Small lesion: Sclerotherapy • Large lesion: Sclerotherapy + surgery 	Combination therapy <ul style="list-style-type: none"> • Embolization (ethanol, coil, or glue) • Surgery for debulking within 48 to 72 hours after embolization to prevent recanalization

Sturge-Weber Syndrome

- Capillary malformation in
 - Ophthalmic and/or maxillary trigeminal nerve distribution
 - Maxillary and/or mandibular malformation
 - Port-wine stain of the face
 - Leptomeningeal system
 - Refractory seizures
 - Hemiplegia
 - Congenital motor and/or cognitive delay
 - Ocular choroid
 - Retinal detachment
 - Glaucoma
 - Blindness

Management

- Face: Multiple pulsed dye laser therapies
- Malocclusion and/or asymmetry: Orthognathic surgery
- Seizure: Anticonvulsant
- Glaucoma: Latanoprost to decrease intraocular pressure

Osteomyelitis

Pathogenesis

- Seeding of bacteria and/or acute inflammation
- Increased intramedullary pressure
- Vascular compromise
- Avascular bone

Risk factors

- Mandible
- Medical conditions
 - Vascular insufficiency
 - Immune dysfunction
 - Metabolic abnormalities (ie, diabetes, Paget disease)
 - Bone pathology (fibro-osseous disease)

Classification

- Time
 - Acute (< 1 month)
 - Chronic (> 1 month)
- Clinical
 - Suppurative
 - Nonsuppurative

Clinical Presentation

Varies based on acuity of the disease

- Acute: Pain (deep and boring), swelling, trismus, paresthesia, fever, and malaise
- Chronic: Mild or asymptomatic

Laboratory tests exhibit normal or mildly elevated white blood cells and elevated protein C and erythrocyte sedimentation rate.

Microbiology

- Difficult to obtain good specimen due to contamination; may require multiple cultures
- *Streptococcus* species, *Staphylococcus aureus*, and *Staphylococcus epidermidis* account for 80% to 90%
- *Escherichia coli*, *Actinomyces*, hemolytic *Streptococcus*, *Eikenella corrodens* account for the remaining 10% to 20%

Imaging

Bony destruction at 30% to 40% is required before radiographic findings are visible (> 4 weeks).

Panoramic radiograph

Good for screening purposes

- “Moth-eaten” appearance
- Island of dead bone (sequestrum)

Bone scintigraphy

High sensitivity but moderate specificity; a negative result means osteomyelitis is unlikely.

- Technetium-99m – Sensitive for increased osteoblastic activity and blood flow – Three phases
 - First: Flow study (1–2 minutes after injection); shows perfusion of lesion
 - Second: Blood pool study (5–10 minutes after injection); shows the relative vascularity of area
 - Third: Delayed study (2–4 hours after injection); shows amount of bone turnover associated with lesion
- Cellulitis: Increases in first two phases but normal in third phase
- Osteomyelitis: Increases in all three phases
- Chronic and/or well-healed osteomyelitis: Mildly abnormal in first two phases but increased uptake in third phase
- Techniques to increase specificity
 - Gallium contrast: Increased uptake due to leaky capillaries in the inflamed site
 - Indium-111: Radioisotope for labeling white blood cells

CT scan and MRI

Greater sensitivity compared to conventional radiograph but does not detect abnormality as early as bone scintigraphy. Useful in surgical treatment planning

- Increased attenuation of medullary cavity
- Destruction of cortical bone
- Presence of sequestrum

Treatment Principles

- Correction of host immune system
- Elimination of infection sources (ie, odontogenic infection and infected necrotic bone)
 - Sequestrectomy and/or saucerization and/or decortication: For small- to moderate-sized defects
 - Resection: For moderate to large defects; healthy bone margin of 1–2 cm
 - Fixation may be needed to promote bony healing or prevent pathologic fracture
 - Maxillomandibular fixation is recommended over internal fixation
 - Preoperative hyperbaric oxygen (HBO) therapy may be used in long-standing refractory cases
 - 20 dives before surgery followed by 10 dives postsurgery
- Establishment of surgical drainage
- Bacteriologic identification and/or long-term antibiotic therapy (~6 weeks)
 - Initiate empiric antibiotic (ie, penicillin or clindamycin); adjust after culture speciation
- Reconstruction

Suppurative Osteomyelitis

Acute suppurative osteomyelitis

- Duration is < 1 month
- Most patients present with moderate to severe symptoms
- Inadequate treatment can lead to chronic disease

Chronic suppurative osteomyelitis

- Duration is > 1 month
- Symptoms are more mild compared to acute pathology
- In late stage can develop orocutaneous fistula and pathologic fracture

Nonsuppurative Osteomyelitis

Chronic nonsuppurative osteomyelitis

- Overgrowth of *Actinomyces* and *Eikenella* species in medullary cavity
- Mild symptoms or asymptomatic
- Can be confused with fibro-osseous lesion

Sclerosing osteomyelitis

- Painful disease with protracted course; intermittent swelling
- Adult 3rd decade of life
- Two types – Diffuse sclerosing osteomyelitis
 - Diffuse intramedullary sclerosis with poorly defined margins
 - Treatment: Long-term antibiotics and surgery
- Focal sclerosing osteomyelitis
 - Localized area of bone sclerosis associated with the apex of a carious tooth and periapical periodontitis
 - Endodontic treatment and/or extraction of affected tooth

- Synovitis acne pustulosis hyperostosis osteitis (SAPHO) syndrome
 - Chronic recurrent multifocal osteomyelitis (CRMO)
 - Spectrum of SAPHO syndrome without cutaneous lesions
 - Auto-inflammatory disease
 - **Age:** Children from 4 to 14 years
 - **Treatment:** Nonsteroidal anti-inflammatory drugs (NSAIDs)

Proliferative periostitis (Garré osteomyelitis)

- Low-grade infection; chronic osteomyelitis with subperiosteal bone and collagen deposition
- Expansile proliferative condition produces onion skin radiographic appearance
- Affects children and young adults
- Treated by removal of source of infection by extraction or endodontic therapy

Osteoradionecrosis

Irradiated bone becomes exposed through a wound in the overlying skin or mucosa causing persistent infection.

Theory	Mechanism	Treatment regimen
Meyer theory (1970s)	<ul style="list-style-type: none"> • Triad: Radiation, trauma (extraction), infection • Extraction leads to increased bacterial access to the underlying bone • Radiation prevents tissue from resisting infection 	Long-term antibiotics
Marx theory (1980s)	<ul style="list-style-type: none"> • Three Hs: Hypocellularity, hypovascularity, and hypoxia • Tissue necrosis (hypoxia) outpaces wound healing 	HBO and surgery
Delanian radiation-induced fibroatrophic theory (2000s)	<ul style="list-style-type: none"> • Activation and/or deregulation of fibroblastic activity (due to free radical production from radiation) • Decreased osteoblastic activity 	Potentiation by clodronate (pentoclo) <ul style="list-style-type: none"> • Pentoxifyline • Tocophenol • Clodronate

Classification

Stage I	<ul style="list-style-type: none"> • Superficial involvement, only cortical bone exposed • Minimal soft tissue ulceration
Stage II A: Minimal soft tissue ulceration B: Soft tissue necrosis	<ul style="list-style-type: none"> • Localized involvement of mandible • Exposed cortical and medullary bone is necrotic
Stage III A: Minimal soft tissue ulceration B: Soft tissue necrosis	<ul style="list-style-type: none"> • Diffuse involvement of the mandible, including the lower border • Pathologic fracture may occur • Possible orocutaneous fistula

Risk factors

Mandible, > 60 Gy radiation, poor oral hygiene, poor nutrition, continued use of ethanol and tobacco

Treatment

- Combination therapy based on different theories
- Prophylactic therapy
 - Extraction of all necessary teeth before radiation or within 5 weeks after radiation
 - After radiation: 20 dives HBO, followed by dental extraction, and 10 dives postextraction
- Early stage: Conservative management with long-term antibiotics, superficial debridement, and observation
- Refractory case
 - Pentoxifylline-tocopherol-clodronate (**PENTOCLO**) therapy
 - Combination of surgical resection and HBO therapy (30 dives before and 10 dives after resection)
- Advanced stage
 - Combination therapy
 - HBO therapy; 30 dives before and 10 dives after surgical resection
 - Soft tissue coverage; free flap or regional flap

Medication-Related Osteonecrosis of the Jaws (MRONJ)

Diagnostic criteria

- History of exposure to bisphosphonates, denosumab (an inhibitor of receptor activator of nuclear factor kappa-B ligand [RANKL]); or antiangiogenic medications
- No history of radiation
- Exposed bone of the jaw for > 8 weeks

Pathogenesis

Bisphosphonates may impair wound healing by:

- Bone remodeling suppression
- Antiangiogenesis
- Local mucosal toxicity (inhibiting fibroblastic proliferation)

Risk factors

- Potency of the drug; risk from intravenous form (2% to 5%) is greater than from oral form (0.04%); zoledronate is greater than pamidronate
- Duration of drug use
- Recent dentoalveolar surgery
- Mandible > maxilla
- Concomitant steroid therapy
- Medical compromise (diabetes)

Classification and treatment

	Description	Treatment
At risk	History of antiresorptive or antiangiogenic drug use (eg, bisphosphonates, denosumab) but no exposed bone	Observation, pain medication and antibiotics, if needed
Stage 0	Nonspecific symptoms, no exposed bone	
Stage 1	Exposed necrotic bone but asymptomatic and free of infection	<ul style="list-style-type: none">• Follow-up in 6 to 8 weeks• Chlorhexidine mouthrinses• Observation or resection
Stage 2	Exposed necrotic bone with symptoms and signs of infection	<ul style="list-style-type: none">• Oral antibiotic (amoxicillin or clindamycin)• Chlorhexidine mouthrinses• Pain medication• Resection
Stage 3	Exposed bone with one of the following findings <ul style="list-style-type: none">• Pathologic fracture• Orocutaneous fistula• Involvement of the inferior border of the mandible or maxillary sinus floor	<ul style="list-style-type: none">• Oral antibiotic (amoxicillin or clindamycin)• Chlorhexidine mouthrinses• Pain medication• Resection

Pediatric Pathology

Jaw Tumors

Odontogenic	Nonodontogenic	
	Benign	Malignant
<ul style="list-style-type: none">• Ameloblastoma (unicystic)• AOT• Ameloblastic fibroma• Odontoma• COT	<ul style="list-style-type: none">• Neurogenic tumor – Neurofibroma/schwannoma<ul style="list-style-type: none">– Melanotic neuroectodermal tumor of infancy• Giant cell tumor• Fibro-osseous lesion• Langerhans cell histiocytosis• Desmoplastic fibroma• Vascular lesion<ul style="list-style-type: none">– Central vascular malformation (may be associated with Osler-Weber-Rendu syndrome)– Intrabony hemangioma (rare)	<ul style="list-style-type: none">• Osteosarcoma• Ewing sarcoma• Lymphoma (non-Hodgkin)<ul style="list-style-type: none">– African subtype

Salivary Gland Diseases

Non-neoplastic	Neoplastic
<ul style="list-style-type: none"> • Mumps • Juvenile recurrent parotitis 	<ul style="list-style-type: none"> • Benign <ul style="list-style-type: none"> – Pleomorphic adenoma • Malignant <ul style="list-style-type: none"> – Low-grade mucoepidermoid carcinoma – Acinic cell carcinoma

Soft tissue lesions

Non-neoplastic	Neoplastic
<ul style="list-style-type: none"> • Infectious etiology <ul style="list-style-type: none"> – Hand-foot-mouth disease – Herpangina – Papilloma – HSV • Non-infectious etiology <ul style="list-style-type: none"> – Eruption cyst – Pyogenic granuloma – Aphthous ulcer – Leukemia <ul style="list-style-type: none"> ◦ Acute myeloid leukemia (AML) gingival hyperplasia 	<ul style="list-style-type: none"> • Benign <ul style="list-style-type: none"> – Aggressive fibromatosis – Vascular anomalies • Malignant <ul style="list-style-type: none"> – Rhabdomyosarcoma

Cutaneous lesions

Infectious	Noninfectious
<ul style="list-style-type: none"> • <i>Molluscum contagiosum</i> (poxvirus) • Impetigo (<i>S aureus</i> or <i>Streptococcus pyogenes</i>) • Warts (HPV 2, 3,4 and 10) 	<ul style="list-style-type: none"> • Acne vulgaris • Epidermal inclusion cyst (sebaceous cyst) • Dermoid cyst • Pilomatrixoma (calcifying epithelioma of Malherbe) <ul style="list-style-type: none"> – Benign skin neoplasm arising from the outer root sheath of hair follicles – Multiple lesions; may be associated with Gardner syndrome – Surgical excision; low recurrence rate • Nevus

Pathology Correlations

Clinical Correlations

	Description	Pathology
Leser-Trélat sign	Sudden onset of multiple (hundreds) of seborrheic keratoses	Sign of internal malignancy
Hutchinson sign	Periungual extension of brown-black pigmentation from longitudinal melanonychia onto the proximal and lateral nail folds	Subungual melanoma
Café au lait spots	Brownish skin lesions	<ul style="list-style-type: none">• Neurofibromatosis type I (coast of California—smooth border)• McCune Albright syndrome (coast of Maine—irregular border)• Gorlin syndrome• Tuberous sclerosis
Neurotropism	Propensity of tumor to invade neural tissue	<ul style="list-style-type: none">• Adenoid cystic carcinoma• Polymorphous low-grade adenocarcinoma
Fontaine sign	Tumor mobile in the lateral plane but not craniocaudally	Carotid body tumor
Nikolsky sign	Slight rubbing of the skin or mucosa results in exfoliation of the outermost layer	<ul style="list-style-type: none">• Pemphigus• Oral lichen planus• Epidermolysis bullosa• Systemic lupus erythematosus• Linear IgA disease• Graft versus host disease• Toxic epidermal necrolysis
Port-wine stain	Pink capillary malformation in the skin	Sturge-Weber syndrome

Histologic Correlations

	Description	Pathology
Fish-net pattern	Description of a plexiform (weblike) histologic appearance	<ul style="list-style-type: none">• Plexiform ameloblastoma• Plexiform neurofibroma
Liesegang rings	Psammomalike calcifications consisting of a dense amorphous core surrounded by concentric layers of precipitate	Pindborg tumor

(Histologic Correlations cont)

	Description	Pathology
Clear cells	A cell containing abundant glycogen or other material that is not stained by hematoxylin and eosin so that the cytoplasm appears clear histologically	<ul style="list-style-type: none"> • Clear cell odontogenic carcinoma • Pindborg tumor • Mucoepidermoid carcinoma • Renal cell carcinoma
Ghost cells	Enlarged eosinophilic epithelial cells with eosinophilic cytoplasm but without a nucleus	<ul style="list-style-type: none"> • Ameloblastoma • Ghost cell odontogenic carcinoma • Gorlin cyst • Odontoma • AFO • Craniopharyngioma
Perivascular hyalinosis	Perivascular collagen deposition	Cherubism
Birbeck granules	Rod-shaped cytoplasmic bodies that have a striated appearance	Langerhans cell histiocytosis
Antoni A and Antoni B	<ul style="list-style-type: none"> • Antoni A: Compact groups of spindle cells with nuclei that tend to show palisading • Antoni B: Loose reticular tissue 	Schwannoma
Verocay bodies	Hyalinized acellular areas composed of reduplicated basement membrane outlined by opposing rows of parallel nuclei	Schwannoma
Herringbone pattern	Cellular pattern resembling a fish skeleton	Fibrosarcoma, malignant peripheral nerve sheath tumor
Starry-sky pattern	Scattered macrophages containing bodies of apoptotic tumor cells	Burkitt lymphoma
Swiss-cheese pattern	Multiple sharply demarcated, variably sized, and randomly scattered rounded spaces superimposed on a relatively homogenous or solid background	Cribriform subtype of adenoid cystic carcinoma
Indian-file pattern	Cells singly dispersed in a line throughout fibrous stroma	Linear subtype of polymorphous low-grade adenocarcinoma
Eosinophilic polygonal cell with swollen cytoplasm	Cell contains abundance of mitochondria in the cytoplasm	Oncocytoma

Radiologic Correlations

Presentation	Pathology
Soap bubble appearance	Ameloblastoma
Honeycomb appearance	Ameloblastoma
Snowflakes	Pindborg tumor
Ground-glass appearance	<ul style="list-style-type: none">Fibrous dysplasiaHyperparathyroidism
Teeth floating in air	<ul style="list-style-type: none">Langerhans cell histiocytosisOsteosarcoma
Sunburst appearance	<ul style="list-style-type: none">OsteosarcomaChondrosarcomaIntraosseous hemangioma
Garrington sign*	<ul style="list-style-type: none">OsteosarcomaChondrosarcoma
Codman triangle†	Osteosarcoma
Punched-out lesions	Multiple myeloma
Cobweb trabeculation	Odontogenic myxoma
Cotton wool appearance	<ul style="list-style-type: none">Paget diseaseCemento-osseous dysplasiaGardner syndromeGigantiform cementoma
Onion skin appearance	<ul style="list-style-type: none">Proliferative periostitisEwing sarcomaLangerhans cell histiocytosis
*Widening of the periodontal ligament space around the affected teeth due to tumor	
†Triangular area of new subperiosteal bone created when a lesion, often a tumor, raises the periosteum	

Correlations Between Syndrome/Disease and Lesions

	Pathology
McCune-Albright syndrome	Triad: Polyostotic fibrous dysplasia, café-au-lait spots, precocious puberty
Hand-Schüller-Christian disease	<ul style="list-style-type: none">Systemic Langerhans cell histiocytosisTriad: Diabetes insipidus, lytic bone lesions, exophthalmos

(Correlations Between Syndrome/Disease and Lesions cont)

	Pathology
Letterer-Siwe disease	Systemic Langerhans cell histiocytosis in neonate
Von Recklinghausen disease	Neurofibromatosis type 1
Wermer syndrome	MEN type 1: Pituitary tumor, parathyroid tumor, pancreatic endocrine tumor
Siipple syndrome	MEN type 2A: Medullary thyroid cancer, pheochromocytoma, parathyroid tumor
Gardner syndrome	Multiple osteomas and intestinal polyps (malignant potential)
Gorlin syndrome	Nevoid basal cell carcinoma syndrome; multiple odontogenic keratocysts, basal cell carcinomas
Triton tumor	MPNST subtype: Malignant schwannoma cells with rhabdomyoblastic differentiation
Heerfordt syndrome	Manifestation of sarcoidosis; triad: uveitis, parotitis, facial nerve paralysis
Bowen disease	Skin SCC in situ
PHACE syndrome	P: Posterior fossa brain malformation H: Hemangioma A: Arterial cerebrovascular anomalies C: Coarctation of the aorta E: Eye and/or endocrine disorder
Sturge-Weber syndrome	Capillary malformation that is characterized by facial port-wine stain and abnormal vessel growth in brain and eye
Marjolin ulcer	Skin SCC arising in burn and/or irradiated area
Trotter syndrome	<ul style="list-style-type: none"> • Associated with advanced nasopharyngeal carcinoma • Features <ul style="list-style-type: none"> – Unilateral conductive deafness – Trigeminal neuralgia – Soft palate immobility – Difficulty opening mouth
Kasabach-Merritt syndrome	Vascular tumor (hemangioma) that leads to decreased platelet count and bleeding problems
Melkersson-Rosenthal syndrome	Neurologic disorder characterized by recurring facial paralysis, swelling of the face and upper lips

Recommended Readings

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EBSCOhost®

Maxillofacial Reconstruction

Din Lam and Andrew Salama

- ▶ Basic Wound Healing
- ▶ Local Flaps
- ▶ Regional Flaps
- ▶ Free Flaps
- ▶ Common Soft Tissue Free Flaps
- ▶ Common Composite Free Flaps
- ▶ Anatomically Oriented Defect Reconstruction
- ▶ Autogenous Bone Grafting

Basic Wound Healing

Wound Healing Process

Stage	Beginning	Duration	Comment
Inflammation	Immediately	4–6 days	<ul style="list-style-type: none"> Exposed collagen activates clotting cascade Fibrin clot provides scaffolding and concentration of cytokines and growth factors Activated macrophages <ul style="list-style-type: none"> Mediate fibroblast growth factor (FGF), platelet-derived growth factor (PDGF), transforming growth factor (TGF) Essential for progression to proliferative phase
Proliferation	4–14 days	1–6 weeks	<ul style="list-style-type: none"> Epithelialization, angiogenesis, and provisional matrix formation Collagen production is hallmark Fibroblasts produce granulation tissue; PDGF and epidermal growth factor (EGF) are main signals of type III collagen and/or glycosaminoglycans
Remodeling	Day 8	Day 8 through years	<ul style="list-style-type: none"> Random to organized fibrils Type III collagen replaced by type I Increased wound strength <ul style="list-style-type: none"> Collagen organization Cross-linking of collagen

Factors Influencing Wound Healing

Local factors

Oxygenation	<ul style="list-style-type: none"> Fibroblasts are oxygen sensitive; when oxygen tension < 40 mm Hg, collagen synthesis cannot take place Hypoxia <ul style="list-style-type: none"> Endothelium responds with vasodilation Capillary leak Fibrin deposition Tumor necrosis factor alpha (TNF-alpha) induction and apoptosis Hyperbaric oxygen <ul style="list-style-type: none"> Normal subcutaneous O₂ tension is 30–50 mm Hg Subcutaneous O₂ tension < 30 mm Hg = chronic wound
Tissue edema	<ul style="list-style-type: none"> Increased tissue pressure Compromised tissue perfusion Cell death and tissue ulceration

(Local factors cont)

Infection	<ul style="list-style-type: none">• Decreased tissue oxygen tension and prolonged inflammatory phase• Impaired angiogenesis and epithelialization• Increased collagenase activity
Tissue temperature	<ul style="list-style-type: none">• Wound healing is accelerated at environmental temperatures of 30°C
Radiation	<ul style="list-style-type: none">• Stasis and occlusion of small vessels• Fibrosis and necrosis of capillaries• Decrease in wound tensile strength• Direct, permanent, and adverse effect on fibroblasts

Systemic factors

Systemic disease	<p>Diabetes</p> <ul style="list-style-type: none">• Larger arteries, rather than the arterioles, are typically affected• Increased dermal vascular permeability and pericapillary albumin deposition• Impaired oxygen and nutrient delivery
Medications	<p>Steroids</p> <ul style="list-style-type: none">• Inhibits both macrophages and neutrophils• Interferes with fibrogenesis, angiogenesis, and wound contraction
Smoking	<ul style="list-style-type: none">• Smoke contains high levels of carbon monoxide<ul style="list-style-type: none">– Shifts the oxygen-hemoglobin curve to the left– Decreases tissue oxygen delivery– Nicotine acts via the sympathetic system, resulting in vasoconstriction and limited distal perfusion• Smoking one pack per day = 3× frequency of flap necrosis• Smoking two packs per day = 6× frequency of flap necrosis
Syndromes	<ul style="list-style-type: none">• Cutis laxa<ul style="list-style-type: none">– Defective elastin fibers– Congenital: Autosomal dominant, recessive, or X-linked recessive– Acquired: Drugs, neoplasms, or inflammatory skin conditions• Ehlers-Danlos syndrome<ul style="list-style-type: none">– Defective collagen metabolism– Autosomal dominant (majority) and recessive patterns –
Hydration	<p>A well-hydrated wound will epithelialize faster than a dry one</p>
Nutrition	<ul style="list-style-type: none">• Low protein levels prolong the inflammation phase• Poor nutrition impairs fibroplasia

Scarring

	Hypertrophic scar	Keloid
Characteristics	<ul style="list-style-type: none">• Not associated with skin pigmentation• Erythematous, pruritic, raised, firm, fibrous lesion• Does not extend beyond the boundary of the scar• Less hereditary related	<ul style="list-style-type: none">• More common in darker skin• Extends beyond original scar boundary• Raised, rubbery lesion• More hereditary related
Location preference	<ul style="list-style-type: none">• Arises in any location• Commonly occurs on extensor surfaces of joints	<ul style="list-style-type: none">• Rarely occurs distal to wrist or knee• Predilection for sternum, mandible, and deltoid region
Histologic differences	<ul style="list-style-type: none">• Absence of hyalinized bundles of collagen• Few thick collagen fibers	<ul style="list-style-type: none">• Haphazard collagen formation• Presence of hyalinized bundles of collagen• Thick collagen fibers
Prognosis	Regresses over time	Grows for years
Management	<ul style="list-style-type: none">• Similar treatment strategy, but keloids are more difficult to eradicate• Combination therapies are usually needed – Corticosteroid injections: 40 mg/cc Kenalog (Bristol-Myers Squibb) intralesional injection (two to three times a month for 6 months)<ul style="list-style-type: none">– Pressure dressing: Silicone dressing worn for 12–24 hours a day for 2–3 months to be effective– Triple therapy<ul style="list-style-type: none">◦ Most effective◦ Surgical excision followed by intralesional steroid injections and silicone dressing• Other treatments: Safe alternatives but low efficiency if used alone<ul style="list-style-type: none">– Intralesional injections<ul style="list-style-type: none">◦ Verapamil for decreased collagen formation◦ Fluorouracil◦ Bleomycin◦ Interferon alpha 2b	

Local Flaps

Flap Classification

- Radom flap: Flap with no named blood vessel supply
- Axial flap: Flap with named blood vessel supply
- Perforator flap: Flap that is based on vessels that perforate and traverse a muscle before piercing the fascia to reach the skin

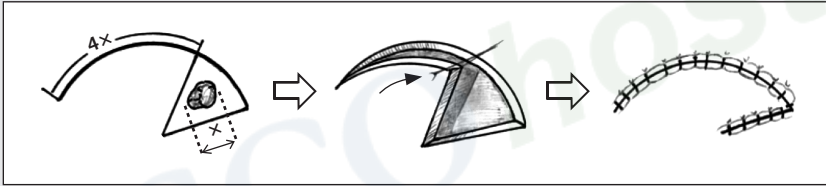
Mathes and Nahai system for muscular flap classification

Type	Description	Examples
Type I	Has only one vascular pedicle	Tensor fascia lata flap
Type II	Has one dominant and one minor pedicle	Gracilis flap
Type III	Has two dominant pedicles	Temporalis flap
Type IV	Has segmental vascular pedicles	Sartorius flap
Type V	Has one dominant pedicle and secondary segmental pedicles	Pectoralis major myocutaneous flap Latissimus dorsi flap

- Most local flaps are randomly based
 - Supplied by subdermal plexus
 - Length:width ratio is 3:1
- Surgical considerations
 - Incisions in relaxed skin tension lines (RSTL)
 - Tension in line of maximal extensibility (LME)
 - Compartmentalize defects crossing facial subunits

Nondistortable landmarks	Tissue sites good for recruitment
<ul style="list-style-type: none">• Hairline• Eyebrow• Eyelid and canthus• Nasal tip and/or ala• Earlobe• Philtrum• Vermilion border• Oral commissure	<ul style="list-style-type: none">• Forehead• Cheek• Chin• Submenton• Neck

Flaps

Advancement flap	<ul style="list-style-type: none"> • Linear movement • Good for forehead and brow defects • Types <ul style="list-style-type: none"> – Primary fusiform closure – Unilateral or bilateral advancement flap – A to T flap – V to Y advancement flap – Island flap
Rotation flap (Fig 8-1)	<ul style="list-style-type: none"> • Pivoted around a fixed point at the base of the flap and rotated along an arc toward the defect • Curvilinear movement (rotate along an arc ≤ 30 degrees) • Radius approximately two to three times the diameter of the defect • Arc length approximately four to five times the width of the defect • Types <ul style="list-style-type: none"> – Scalp (advancement-rotation) – O to Z – Cervicofacial flap  <p>Fig 8-1 Rotation flap. (Reprinted with permission from Patel KG, Sykes JM. Concepts in local flap design and classification. Oper Tech Otolaryngol 2011;22:13–23.)</p>
Transposition flap	<ul style="list-style-type: none"> • Linear axis • No need for second-stage procedure • Types <ul style="list-style-type: none"> – Limberg rhomboid flap (Fig 8-2a) <ul style="list-style-type: none"> ◦ Based on designing a rhombus around the defect with angles of 60 and 120 degrees ◦ Duformental and Webster modifications: Based on creating smaller angles of rotation (less redundancy and smaller standing cone deformities) – Bilobed flap (Fig 8-2b) <ul style="list-style-type: none"> ◦ Double transposition <ul style="list-style-type: none"> – First lobe, same diameter as the defect – – Second lobe, half the diameter of the defect ◦ Arcs of 90 to 110 degrees ◦ Best for defects < 1.5 cm in lower third of nose – Z-plasty <ul style="list-style-type: none"> ◦ Central limb contains the area from which scar tissue is excised ◦ Good for defects that require increased length for closure <ul style="list-style-type: none"> – 30 degrees, increase by 25% – 45 degrees, increase by 50% – 60 degrees, increase by 75%

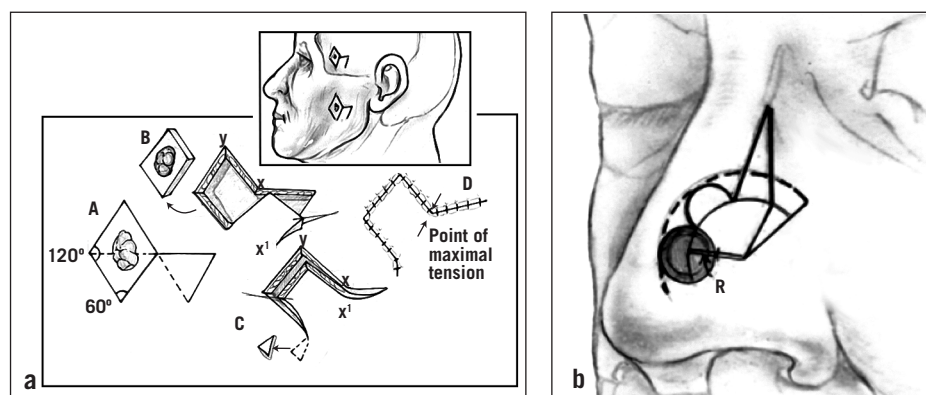


Fig 8-2 (a) Limberg flap. (b) Bilobed flap. R, radius of defect. (Reprinted with permission from Patel KG, Sykes JM. Concepts in local flap design and classification. Oper Tech Otolaryngol 2011;22:13–23.)

Scar Revision

At least 6 months after original treatment.

Nonsurgical management

- Kenalog injection
 - Management of hypertrophic scars and keloids
 - Mechanism: Reduced angiogenesis, fibroblast proliferation, and production of collagen and extracellular matrix protein
 - Can be used at various times during therapy
 - Prophylaxis in early postsurgical treatment: 20 mg/cc
 - Prophylaxis in late postsurgical treatment: 40 mg/cc
 - Treatment: 40 mg/cc with multiple injections given 4–6 weeks apart

Surgical management

- Small (< 2 cm): Single elliptical excision, Z-plasty
- Moderate (2 to 5 cm): Geometric W-plasty
- Large (> 5cm): Serial excision or local flap

Regional Flaps

Head Flaps

Temporoparietal flap (Fig 8-3)

- Based on superficial temporal artery
- Continuous with the superficial muscular aponeurotic system (SMAS) inferiorly and the galea superiorly
- **Indications**
 - Orbital and maxillary reconstruction
 - Auricular reconstruction to cover the reconstructed cartilage
- **Advantages**
 - Thin and pliable tissue
 - Can harvest with a hair-bearing skin paddle
 - Minimal donor site morbidity
- **Disadvantage**
 - Difficulty in dissecting in the superficial plane

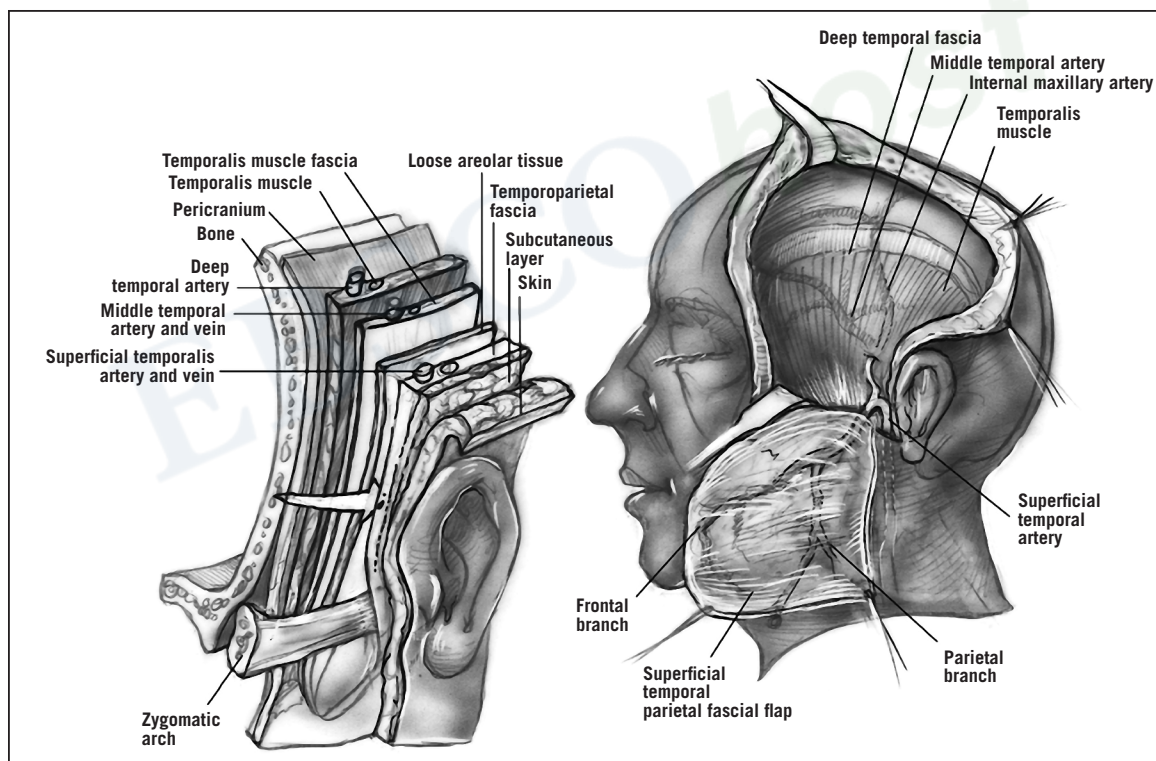


Fig 8-3 Temporoparietal flap. (Reprinted with permission from Chepeda DB, Teknos TN. Microvascular free flaps in head and neck reconstruction. In: Bailey BJ, Johnson JT, Newlands SD [eds]. Head and Neck Surgery—Otolaryngology, vol 2, ed 4. Philadelphia: Lippincott Williams & Wilkins, 2006:2369–2392.)

(Head Flaps cont)

Temporalis flap (see Fig 8-3)	<ul style="list-style-type: none">• Has two components – Anterior component—anterior and posterior deep temporal artery – Posterior component—middle temporal artery (branch of the superficial temporal artery)• Indications<ul style="list-style-type: none">– Obliteration of oral defects (ie, oroantral communication)– Cranial base reconstruction– Facial reanimation surgery– Temporomandibular joint gap arthroplasty– Midface and orbital repair• Advantages<ul style="list-style-type: none">– Excellent bulk for intraoral defect– Easy dissection• Disadvantage – Cosmetic defect—temporal hollowing – Can improve with<ul style="list-style-type: none">◦ Medpor (Stryker) implant◦ Repositioning the posterior flap anteriorly
Paramedian flap	<ul style="list-style-type: none">• Axial flap based on supratrocheal artery (1.7 to 2.2 cm from midline)• Common indication: Large nasal defect• The flap may be extended into the hair-bearing scalp and/or, for additional length, up to 1.5 cm below the orbital rim• Advantages<ul style="list-style-type: none">– Reliable flap– Minimal donor site morbidity– Good tissue match• Disadvantage<ul style="list-style-type: none">– Requires second surgery for pedicle division after 3 weeks

Orofacial Arterial System

Nasolabial flap	<ul style="list-style-type: none">• Can be random or axial flap• Axial flap based on angular artery• Inferior or superior based flap• Indications: Lower 2/3 of nose, perinasal area, upper lip, closure of small- to moderate-sized intraoral palatal defects• Disadvantages<ul style="list-style-type: none">– Blunting of nasofacial sulcus– Potential ectropion– May cause scleral show– Limited arc of rotation
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(Orofacial Arterial System cont)

Facial artery myomucosal (FAMM) flap	<ul style="list-style-type: none"> • Based on branch of facial artery • Inferior based; reconstruction of small to moderate defects in <ul style="list-style-type: none"> – Lower alveolus – Floor of the mouth – Vermilion of the lip • Superior based; reconstruction of palatal and upper alveolar defects • Advantage <ul style="list-style-type: none"> – Similar soft tissue for defect reconstruction • Disadvantages <ul style="list-style-type: none"> – May restrict mouth opening – Limited arc of rotation
Tongue flap	<ul style="list-style-type: none"> • 3–10 mm thickness • Can be a random or axial flap • Random flap – Anterior based for hard palate, anterior buccal mucosa, anterior floor of mouth, and lip <ul style="list-style-type: none"> – Posterior based for soft palate, retromolar region, posterior buccal mucosa – Lateral based for buccal mucosa, lateral palate, and alveolus – Double-door modification for relining large defects of the buccal mucosa • Axial flap <ul style="list-style-type: none"> – Based on dorsal-lingual branch of lingual artery • Disadvantages <ul style="list-style-type: none"> – Requires second surgery for pedicle division – May require revision for debulking (3 months postoperatively)
Palatal island flap	<ul style="list-style-type: none"> • Based on greater palatine artery • Common indications: Palatal defect (oroantral communication) and retromolar region • Can increase pedicle length by fracturing the pterygoid hamulus • Can harvest the entire palatal tissue (across midline) based on single vascular pedicle • Can rotate as much as 180 degrees • Advantage: Minimal donor site morbidity
Submental island flap	<ul style="list-style-type: none"> • Based on submental artery (branch of facial artery) • Incorporates anterior digastric muscle and portion of mylohyoid muscle to protect vascular pedicle • Common indications: Floor of the mouth, tongue, retromolar, and soft palate defects • Advantages <ul style="list-style-type: none"> – Low donor site morbidity – Excellent cutaneous color, texture, and thickness match • Disadvantage <ul style="list-style-type: none"> – May not be applicable in most oral malignancy cases with possible level I lymph node metastasis (except sarcoma and adenoid cystic carcinoma)

Neck Flaps

Cervicofascial flap	<ul style="list-style-type: none">• Random fasciocutaneous flap in the face and a musculocutaneous flap in the neck• Indication: Resurfacing cutaneous defects in the head and neck region• Advantages<ul style="list-style-type: none">– Reliability and ease of harvesting– Best cosmetic match for surface area in the head and neck– Can provide large cutaneous coverage (especially in elderly patients)• Disadvantages – Limited volume; only contains skin and subcutaneous tissue – May not be suitable for patients who had previous neck dissection or radiation therapy
Platysma flap	<ul style="list-style-type: none">• Not a true axial flap with musculocutaneous perforators• Based on<ul style="list-style-type: none">– Submental branch of facial artery– Superior thyroid artery• Indications: Reconstruct defects on the lower face, buccal mucosa, and floor of mouth• Advantages<ul style="list-style-type: none">– Thin and pliable tissue– Minimal donor site morbidity• Disadvantages<ul style="list-style-type: none">– Not suitable in patients who had neck dissection previously or radiation to the neck– Difficult to harvest, especially in thin patients– Limited arc of rotation
Sternocleidomastoid (SCM) flap	<ul style="list-style-type: none">• Only the superior-based SCM flap is useful for head and neck reconstruction• Only use one of the heads (sternal or clavicular) for reconstruction and leave the other to provide protection to the great neck vessels• Based on branches of occipital and superior thyroid arteries• Indications – Lateral and lower face defects – Intraoral defects – Improve facial contour after parotidectomy (also helps to decrease the incidence of Frey syndrome)<ul style="list-style-type: none">– Facial and tongue reanimation• Advantages<ul style="list-style-type: none">– Ease in harvesting– Proximity to the required defect– Minimal donor site morbidity• Disadvantages<ul style="list-style-type: none">– May not be suitable for most oncologic patients– Limited arc of rotation

Transverse Cervical System

- Branch of thyrocervical trunk
- Passes transversely above the inferior belly of the omohyoid muscle
- Two branches
 - Superficial; not commonly used
 - Deep; can sometimes arise from subclavian artery

Trapezius flap	<ul style="list-style-type: none"> • Two types <ul style="list-style-type: none"> – Superior trapezius flap – Lower island trapezius flap • Common indications: Lateral neck and skull cutaneous defects • Arterial supply: Dorsal scapular artery • Advantages <ul style="list-style-type: none"> – Hairless skin – Long, thin pedicle allows easy transfer • Disadvantages <ul style="list-style-type: none"> – Requires patient repositioning – Limited application in oral and maxillofacial region – May not be suitable for patients who had previous neck dissection – Weakening of shoulder function
Supraclavicular flap (Fig 8-4)	<ul style="list-style-type: none"> • Supraclavicular artery <ul style="list-style-type: none"> – 93% arise from transverse cervical artery: 8 cm lateral to the sternoclavicular joint, 3 cm superior to the clavicle, and 2 cm posterior to the SCM – 7% arise from suprascapular artery • Indications – Lower face cutaneous defects – Partial or circumferential pharyngoesophageal reconstruction – Postburn neck scar contracture release and soft tissue ablation, repair of the lip, chin, cheek, and oropharynx • Advantages <ul style="list-style-type: none"> – Ease of harvesting – Low donor site morbidity – Thin, pliable flap • Disadvantages <ul style="list-style-type: none"> – May not be applicable in patients who had previous neck dissection and/or radiation – Potential for distal flap necrosis – Anatomical variations of the supraclavicular artery

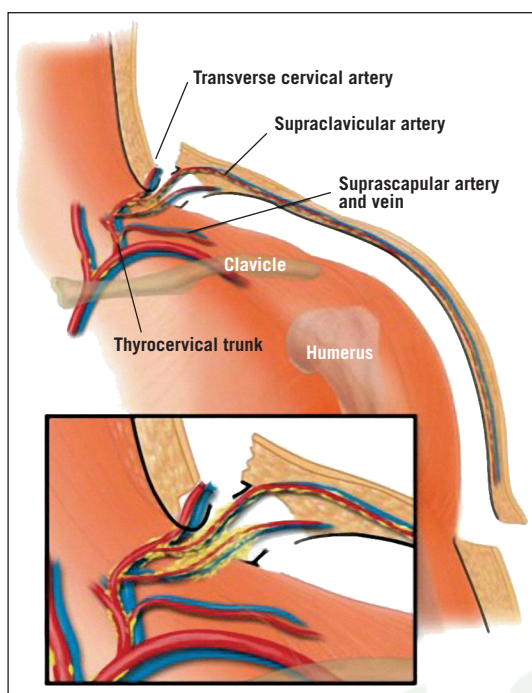


Fig 8-4 Supraclavicular flap. (Reprinted with permission from Granzow JW, Suliman A, Roostaeian J, Perry A, Boyd JB. The supraclavicular artery island flap [SCAIF] for head and neck reconstruction: Surgical technique and refinements. *Otolaryngol Head Neck Surg* 2013;148:933–940.)

Chest Flaps

Pectoralis major myocutaneous flap (PMMF)

- Based on
 - Thoracoacromial artery—dominant
 - Long thoracic artery
- **Indications**
 - Most head and neck defects; can reach as far as the skull base
 - Reserve for large head and neck defects especially when microvascular reconstruction is not feasible
- **Advantages**
 - One-stage reconstruction
 - Patient repositioning is not needed
 - Ease of harvesting
 - Can provide soft tissue for neck vessel protection when reconstructing the head and neck defect
- **Disadvantages**
 - Can alter breast symmetry in women
 - Postoperative decrease in arm and shoulder function

*(Chest Flaps cont)***Deltpectoral flap**

- Based on perforators from internal mammary artery
- **Indication:** Usually reserved as a last option for head and neck defects
- **Advantages**
 - Reliable and provides large soft tissue bulk
 - Easy to harvest
- **Disadvantages**
 - Requires two surgeries separated by 3–6 weeks
 - Limited arc of rotation
 - May not be available if previous PMMF has been done

Free Flaps**Advantages Over Regional Flaps**

- Wide variety of available tissue types
- Large amount of composite tissue (skin, fat, muscle, fascia, and bone)
- Tailored to match defect
- Wide range of skin characteristics

Vessel Suturing Technique

- Average of eight stitches; six stitches if vessel diameter is small
- Mechanical coupler can be used for venous anastomosis; arterial anastomosis carries 25% failure rate
- Vein graft
 - Used in situations where pedicle is not long enough for tension-free arterial anastomosis
 - Saphenous vein is most commonly used
 - Valve orientation is important

Recipient Vessel Selection

- Midface
 - Short pedicle—superficial temporal artery (small diameter)
 - Long pedicle—facial artery/vein
- Lower face and/or neck
 - Artery—facial artery, superior thyroid artery, and transverse facial artery
 - Vein—external/internal jugular

Vessel Discrepancy

- < 2:1—dilation
- > 2:1 or < 3:1—beveling or spatulation (no more than 30 degrees to avoid turbulence)
- > 3:1—end to side anastomosis (less than 60 degrees to minimize turbulence)

Ischemic Time

- Skin and subcutaneous tissue: 4 to 6 hours (warm); up to 12 hours (cold)
- Muscle: < 2 hours (warm); 8 hours (cold)
- Bone: < 3 hours (warm); 24 hours (cold)

Flap Monitoring

- Clinical evaluation: Color, capillary refill, turgor, and temperature—gold standard
- Pinprick: Assess flow and color
- Devices: Doppler signal, tissue pH, and implantable Doppler

	Arterial occlusion	Venous congestion
Flap color	Pale	Cyanotic, bluish color
Capillary refill	Sluggish	Brisk
Tissue turgor	Decreased	Increased
Temperature	Cool	Cool
Pinprick test	Scant amount of dark blood	Rapid bleeding of dark blood
Doppler signals	Absence of pulsatile arterial signals	Absence of continuous venous signals

Factors Associated with Flap Failure

Venous: Arterial thrombosis—4:1

Important factors	Possible factors	Nonfactors
<ul style="list-style-type: none">• Use of vein graft• Pedicle characteristics• Pedicle positioning (kinking)• Surgery time• Tight closure• Presence of infection• Patient factors<ul style="list-style-type: none">– Obesity– Smoking– Peripheral vascular	<ul style="list-style-type: none">• Patient factors<ul style="list-style-type: none">– Age– Low preoperative hemoglobin level– Diabetes• Radiation• Vessel selection (internal jugular versus external jugular)• Location of defect (skull base/midline)• Excess intraoperative fluids > 7 L or 6 cc/kg/h• Osseous containing flaps• Hypothermia	<ul style="list-style-type: none">• Type of anastomosis (end to end versus end to side)• Running sutures versus interrupted sutures versus coupler• Loupes versus microscope• Hypotension and use of vasopressor

Perioperative Anticoagulation Regimens

Agents	Usage	Prevalence of use in US microsurgical practice
Heparin	Variable protocols <ul style="list-style-type: none"> Weight-based dosage Start at the beginning of the case then two to three times a day 	27%
Aspirin	81 mg for 14 days, 325 mg until discharge	77%
Dextran	12 hours on, 12 hours off for 5 days	35%

Common Soft Tissue Free Flaps

Radial Forearm Fasciocutaneous Flap (RFFF)

Type	Septocutaneous flap
Artery	Radial artery
Vein	<ul style="list-style-type: none"> Venae comitantes (deep system) Cephalic vein (superficial system)
Vessel diameter	<ul style="list-style-type: none"> Radial artery: 3.0 mm Cephalic vein: 3.0 mm Venae comitantes: 1.5 mm
Nerve	Lateral antebrachial cutaneous nerve (travels with cephalic vein)
Septum	Lateral intermuscular septum
Muscles/tendon	Radial artery is located between <ul style="list-style-type: none"> Brachioradialis Flexor carpi radialis
Indications	<ul style="list-style-type: none"> For oral and oropharyngeal reconstruction to replace mucosa Tubed flap for pharyngeal and cervical esophageal reconstruction Mandibular reconstruction (nonloaded zone) Skin resurfacing or soft tissue augmentation in the face and neck region
Perioperative considerations	<ul style="list-style-type: none"> Always choose the nondominant hand first Complete preoperative Allen test Use a tourniquet; can be set at 250 mm Hg and maintained for 90 minutes Preserve paratendon to increase success rate of skin graft

(RFFF cont)

Advantages	<ul style="list-style-type: none"> • Two-team approach • Ease of harvesting • Consistent vascular anatomy • Long pedicle and large diameter vessels • Thin, pliable skin flap
Disadvantages	<ul style="list-style-type: none"> • Approximately 10% of patients are not candidates for forearm flap <ul style="list-style-type: none"> – Aplastic radial artery (< 1% associated with Down syndrome) – Inadequate blood flow to hand (10%); only when both of the following conditions co-exist <ul style="list-style-type: none"> ◦ Incomplete superficial palmar arches ◦ Lack of communication between deep and superficial palmar arches • Persistent paresthesia • Poor donor site cosmesis • Limited amount of bone
Postoperative considerations	<ul style="list-style-type: none"> • Monitor hand temperature and capillary refill to avoid compartment syndrome • If skin graft is used, wrist should be held in a flexed position with a back slab plaster for 5–7 days; prevents shearing of the underlying tendon and increases success rate of skin graft • If bone is harvested, arm should be in a cast and immobilized for 3–5 weeks
Modifications	<ul style="list-style-type: none"> • Teninocutaneous flap <ul style="list-style-type: none"> – Incorporation of palmaris longus muscle into the flap – Indicated for lip reconstruction: Tendon serves as a suspension sling • Osteocutaneous flap <ul style="list-style-type: none"> – Incorporation of radial bone into the flap – No more than 25% of radial bone circumference can be harvested – Length of 10–12 cm can be harvested; insertion of the pronator teres to the distal styloid—no muscle attachment – Titanium plate is placed to prevent pathologic fracture of the radius – Best used for nonloading osseous reconstruction (angle of mandible and maxilla)

Anterolateral Thigh (ALT) Flap

Type	<ul style="list-style-type: none"> • Septocutaneous: 12% • Musculocutaneous: More common
Vessels	<ul style="list-style-type: none"> • Skin perforators originate from descending branch of lateral circumflex femoral artery (LCFA) • 20% of skin perforators originate from oblique branch of LCFA • Venae comitantes
Vessel diameter	2-mm diameter
Nerve	Lateral femoral cutaneous nerve
Perforator	<p>Three different patterns</p> <ul style="list-style-type: none"> • Type 1: Most reliable perforator originates from descending branch of LCFA (92%) • Type 2: Most reliable perforator originates from transverse branch of LCFA (4%) • Type 3: Most reliable perforator originates from profundus femoris artery (4%) <ul style="list-style-type: none"> – Usually too small for reconstructive use – Alternative flap, anteromedial thigh, or contralateral thigh should be considered
Muscles	<p>LCFA is located between</p> <ul style="list-style-type: none"> • Rectus femoris • Vastus lateralis, where intramuscular perforators will pierce through
Indications	<ul style="list-style-type: none"> • Oral and oropharyngeal reconstruction to replace mucosa • Tubed flap for pharyngeal and cervical esophageal reconstruction • Mandibular reconstruction (nonloaded zone) • Skin resurfacing or soft tissue augmentation in the face and neck region
Size for primary closure	<ul style="list-style-type: none"> • 8 cm in width • Use split-thickness skin graft (STSG) if defect > 8 cm in width
Indications	<ul style="list-style-type: none"> • Oral and oropharyngeal mucosal reconstruction • Oral commissure: Use tendon fascia lata as a suspension sling • Pharyngeal and cervical esophageal reconstruction; minimal 9 cm in width to create a 3-cm-diameter tube • Skull base reconstruction • Skin resurfacing or augmentation in head and neck region

(ALT Flap cont)

Preoperative examination	<ul style="list-style-type: none"> • Check for previous scars in the region • Check for knee extension and stability <ul style="list-style-type: none"> – Trauma to vastus lateralis during flap harvesting can further impair knee function • Patient body habitus <ul style="list-style-type: none"> – Obese patients may require flap thinning – Men may have thick hair that requires laser hair removal – Westerner flap thickness is 2× the easterner flap • Preoperative perforator identification – Handheld Doppler; decreased sensitivity with increased patient body mass index (BMI)
Surface anatomy	<ul style="list-style-type: none"> • Anterior superior iliac spine (ASIS) to lateral superior corner of patella (AP line—the imaginary line for the septum between the vastus lateralis and rectus femoris) • Highest concentration of perforators located within the 3-cm-diameter circle that is located midway between ASIS and patella (AP line) • Three commonly found perforators (ABC system) <ul style="list-style-type: none"> – Point A: 5 cm proximal to the midpoint of the AP line (presence in 49%) – Point B: Midpoint of the AP line (presence in 93%) – Point C: 5 cm distal to the midpoint of the AP line (presence in 63%)
Advantages	<ul style="list-style-type: none"> • Two-team approach • Ease of harvesting • Versatility in design, with variable thickness • Vascular pedicle is relatively arteriosclerotic free • Long vascular pedicle (12 cm) • Ability to create chimeric flap
Disadvantages	<ul style="list-style-type: none"> • Color mismatch compared to the facial skin • Presence of hair in some men • Postoperative donor-site paresthesia is common • Excessive thickness in some patients
Postoperative management	<ul style="list-style-type: none"> • Monitor for dorsalis pulse and temperature to prevent compartment syndrome in lower extremity • Bed rest is recommended for 7 days if skin grafting is done
Modifications	<ul style="list-style-type: none"> • Suprafascial versus subfascial dissection <ul style="list-style-type: none"> – Suprafascial dissection allows thinning of the flap but is technically more demanding – Subfascia is easier to dissect • Adipofascial flap <ul style="list-style-type: none"> – Flap is harvested without the skin paddle (fat and fascia only) • Innervated flap <ul style="list-style-type: none"> – Lateral femoral cutaneous nerve (sensory); 10 cm distal to the ASIS – Inclusion of the motor nerve to the vastus lateralis for muscular functional flap • Chimeric flap <ul style="list-style-type: none"> – Two independent paddles based on separate perforators but originating from the same vessel

Other Commonly Used Soft Tissue Flaps

	Vessel	Advantages	Disadvantages	Comments
Latissimus dorsi	Thoracodorsal artery	<ul style="list-style-type: none"> Provides wide soft tissue coverage Hairless skin Wide vessel diameter 	Requires patient repositioning (lateral decubitus position)	<ul style="list-style-type: none"> Large soft tissue defects <ul style="list-style-type: none"> Through and through defect Skull base defect Can be incorporated with scapular tip
Ulnar flap	Ulnar artery +/- basilic vein	<ul style="list-style-type: none"> Thin, pliable flap Less hirsute skin Less cold intolerance and better donor site cosmesis compared to RFFF 	<ul style="list-style-type: none"> Dissection can lead to ulnar nerve injury Pedicle length is shorter than RFFF (10 cm) 	<ul style="list-style-type: none"> Good alternative to RFFF Preoperative Allen test required Can also incorporate palmaris longus for lip reconstruction or medial cutaneous nerve for sensate flap
Lateral arm flap	Posterior radial collateral artery (terminal branch of profunda brachii artery)	<ul style="list-style-type: none"> Great variability in flap design Consistent vascular anatomy 	<ul style="list-style-type: none"> Radial nerve damage Small vessel diameter possible (< 1 mm) 	Most head and neck reconstructions are possible
Rectus flap	Deep inferior epigastric artery/vein OR Deep superior epigastric artery/vein	<ul style="list-style-type: none"> Consistent vascular anatomy Two-team approach Good donor site esthetics Versatility in soft tissue design (transverse, vertical, or oblique) 	<ul style="list-style-type: none"> Weakening of the abdominal wall Flap shrinkage over time due to muscle atrophy 	<ul style="list-style-type: none"> Head and neck reconstruction that requires soft tissue bulk Tongue reconstruction

RFFF, radial forearm fasciocutaneous flap.

Common Composite Free Flaps

Fibular Flap (Fig 8-5)

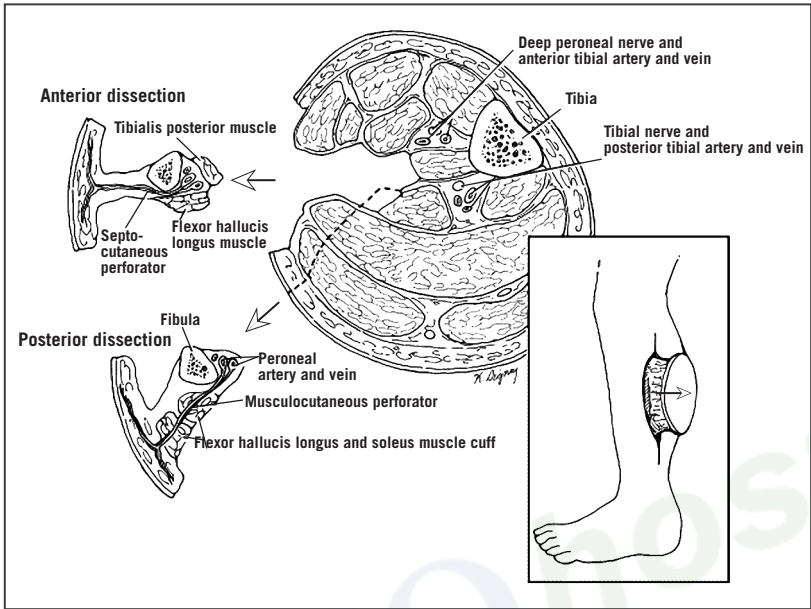


Fig 8-5 Fibular flap. (Reprinted from Blanchert RH Jr, Harris CM. Microvascular free bone flaps. Atlas Oral Maxillofac Surg Clin North Am 2005;13:151–171.)

Type	<ul style="list-style-type: none">• Osseous flap• Composite flap<ul style="list-style-type: none">– Osteoseptocutaneous flap; more common when the skin paddle is in the middle and distal third of the fibula– Osteomusculocutaneous flap; more common when the skin paddle is in the proximal third of the fibula
Artery	<ul style="list-style-type: none">• Peroneal artery• Variations<ul style="list-style-type: none">– Arises from posterior tibial artery (90%)– Arises from anterior tibial artery (1%)– Arises from popliteal artery (1%)– Peronea magna—takes the place of the anterior and posterior tibial artery (8%)<ul style="list-style-type: none">◦ Contraindicated in fibular harvesting
Vein	Venae comitantes
Vessel diameter	1.5–3 mm
Nerve	Lateral sural cutaneous nerve
Muscles	<ul style="list-style-type: none">• Flexor hallucis longus• Soleus muscle

(Fibular Flap cont)

Septum	Posterior crural septum
Size for primary closure	<ul style="list-style-type: none"> Primary closure if width of skin paddle is < 4 cm STSG for width > 4 cm
Common indications	<ul style="list-style-type: none"> Mandibular reconstruction James Brown type III or IV maxillary defect (see page 307)
Extremity selection (Fig 8-6)	<ul style="list-style-type: none"> Location of the anastomosis: Anterior versus posterior Skin paddle placement: Intraoral versus extraoral <div data-bbox="626 615 1411 972"> <pre> graph TD A[Donor site selection] --> B[Where is the defect?] B --> C[Internal (oral cavity)] B --> D[External (skin)] C --> E[Anterior] C --> F[Posterior] D --> G[Anterior] D --> H[Posterior] E --> I[Ipsilateral] F --> J[Contralateral] G --> K[Contralateral] H --> L[Ipsilateral] </pre> </div> <p>Fig 8-6 Algorithm for choosing fibular donor site. (Reprinted with permission from Yagi S, Kamei Y, Torii S. Donor side selection in mandibular reconstruction using a free fibular osteocutaneous flap. <i>Ann Plast Surg</i> 2006;56:622–627.)</p>
Preoperative examination	<ul style="list-style-type: none"> Preoperative magnetic resonance arteriogram or duplex ultrasonography may be warranted in ruling out peronea magna Caution in patients with previous history of <ul style="list-style-type: none"> Peripheral vascular disease Deep vein thrombosis Trauma
Surgical tips	<ul style="list-style-type: none"> Tourniquet is used around the thigh and inflated to 325–375 mm Hg (for no longer than 90 minutes) Proximal and distal 8 cm of fibula needs to be preserved to prevent knee and ankle instability Avoid injury to the superficial peroneal nerve If no septocutaneous perforators are found in the posterior crural septum, need to harvest portion of flexor hallucis longus and soleus muscles Closure management – Flexor hallucis longus is sutured to tibialis posterior muscle and to intraosseous membrane <ul style="list-style-type: none"> Foot is placed in 90 degrees of dorsiflexion, and the great toe is positioned in slight hyperextension during closure Skin graft if skin paddle > 4 cm has been harvested

(Fibular Flap cont)

Postoperative management	<ul style="list-style-type: none"> • Posterior splint that extends beyond the first toe should be applied; avoids flexion contracture that may limit ankle and toe function • Monitor for compartment syndrome if primary closure is done • Ambulation should not start until 2 weeks after surgery
Postoperative complications	<ul style="list-style-type: none"> • Weakness in dorsiflexion of great toe <ul style="list-style-type: none"> – Injury to peroneal nerve – Scarring of the flexor hallucis longus • Gait disturbance—negative impact when performing complicated action or in high velocity • Equinovarus deformity—injury to common peroneal nerve

Comparison of Composite Free Flaps

	Fibular	Iliac crest	Scapular
Artery	Peroneal artery	Deep circumflex iliac artery (DCIA)	Circumflex scapular artery <ul style="list-style-type: none"> • Horizontal • Vertical (parascapular)
Vessel diameter	1.0–2.5 mm	2–3 mm	2–2.5 mm
Indication	Composite defects > 9 cm in length	Composite defects < 9 cm in length	Composite defects with large soft tissue defects
Bone height for dental implants	< 10 mm in height	> 10 mm in height	Insufficient
Advantages	<ul style="list-style-type: none"> • Longest segment of revascularized bone (25 cm) • Only osseous flap available for total mandibular reconstruction • Allows multiple osteotomies (minimal 3 cm per segment) 	<ul style="list-style-type: none"> • 16 cm of bone available • Two-team approach is possible • Allows two independent soft tissue paddles <ul style="list-style-type: none"> – Skin paddles (skin perforators from DCIA) – Internal oblique muscle (ascending branch of DCIA) 	<ul style="list-style-type: none"> • Versatility in soft tissue design <ul style="list-style-type: none"> – Two separate skin paddles – Horizontally oriented flap—transverse cutaneous branch – Vertically oriented flap, parascapular flap—descending cutaneous branch • Skin is thin and hairless • 10 cm of bone length available

(Comparison of Composite Free Flaps cont)

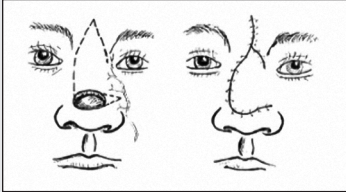
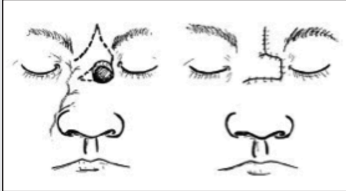

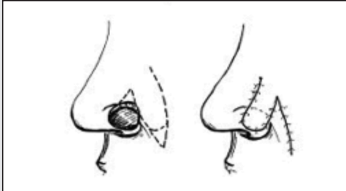
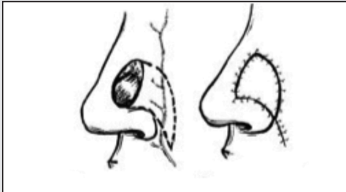
Disadvantages	<ul style="list-style-type: none">• Obvious donor site scar• Limitations and discomfort in ankle function and range of motion with aggressive physical activity (15% to 30%)	<ul style="list-style-type: none">• Variable vascular anatomy• Difficulty harvesting in obese patients• Risk of abdominal hernia• Moderate pedicle length• Gait disturbance• Skin paddle is too thick for oral lining	<ul style="list-style-type: none">• Bone stock for dental implants is limited• Limited osteotomies can be done with the harvested bone• Requires patient repositioning (single-team approach)• No sensory reinnervation
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Anatomically Oriented Defect Reconstruction

Nose Reconstruction

- Skin in lower $\frac{2}{3}$ is thick and sebaceous; in upper $\frac{1}{3}$, it is thin and transparent
- Tissue consists of three layers: Skin, cartilage (bone), and mucosal lining
- Selection of treatment should be based on size, depth (layer), orientation, and location of defect
- Location can be classified into
 - Proximal $\frac{1}{3}$
 - Full-thickness defect that usually does not lead to nasal collapse
 - Bony and nasal lining reconstruction are usually not required
 - Middle $\frac{1}{3}$
 - Full-thickness defect requires reconstruction of nasal lining and underlying cartilage
 - Distal $\frac{1}{3}$
 - Most common location requiring reconstruction
 - Can separate site into six individual units: (1) alar, (2) domal-alar groove, (3) dome, (4) central tip, (5) columella, and (6) nasal sill
 - Full-thickness defect requires both cartilage and nasal lining
- If defect is greater than 50% of any subunit, the entire subunit should be excised and reconstructed

External skin reconstruction options

Miter flap (Fig 8-7*)	<ul style="list-style-type: none"> • Advancement flap • Recruits tissue vertically 	
Glabella flap (Fig 8-8*)	<ul style="list-style-type: none"> • Transposition flap • Recruits tissue from glabella region 	
Bilobed flap (Fig 8-9*)	<ul style="list-style-type: none"> • Transposition flap • Ideal for dome or central tip defect 	
Nasolabial flap (Fig 8-10*)	<ul style="list-style-type: none"> • Transposition flap (angular artery) • Superiorly or inferiorly based • Requires second-stage procedure most of the time 	
V to Y flap (Fig 8-11*)	<ul style="list-style-type: none"> • Advancement flap • Ideal for lateral defects of proximal or middle 1/3 of the nose 	
Paramedian flap	<ul style="list-style-type: none"> • Transposition flap (supratrochlear artery) • Excellent color match • Ideal for total/subunit reconstruction 	

*Figs 8-7 to 8-11 modified with permission from Parrett BM, Pribaz JJ. An algorithm for treatment of nasal defects. Clin Plast Surg 2009;36:407–420.

Cartilage and bone donor sites

Cartilage reconstruction can be done

- At the same time as the skin and nasal lining
- At 3 weeks after soft tissue reconstruction

Septal cartilage	<ul style="list-style-type: none">• Best available cartilage, similar to the nasal cartilage• Limited in quantity
Conchal cartilage	<ul style="list-style-type: none">• Weaker cartilage• Ideal material for alar reconstruction because of intrinsic curve
Costal cartilage	<ul style="list-style-type: none">• Strong cartilage, but with enhanced morbidity associated with harvesting• Sixth to ninth ribs used most commonly
Cranial bone	<ul style="list-style-type: none">• Great for proximal third reconstruction (bony pyramid and lateral wall)• Harvest from the parietal skull• Microplate required for fixation

Nasal lining reconstruction

Septal mucoperichondrial flap	<ul style="list-style-type: none">• Most commonly used• Can be harvested together with septal cartilage• Based on septal artery (branch of superior labial artery)
FAMM flap	<ul style="list-style-type: none">• Superiorly based flap (facial artery) from intraoral buccal mucosa• Requires tunneling from oral cavity to reach to the recipient site
Full-thickness skin graft (FTSG)	Requires vascularized recipient bed

Nose reconstruction algorithm

Nasal division	Subdivision	Orientation	Flap choice
Proximal third	Central	Horizontal	Miter flap
		Round	Glabella flap
		Vertical	V to Y flap
	Lateral	Horizontal	Glabella flap, FTSG
		Vertical	V to Y flap, FTSG
	Combined		Paramedian flap

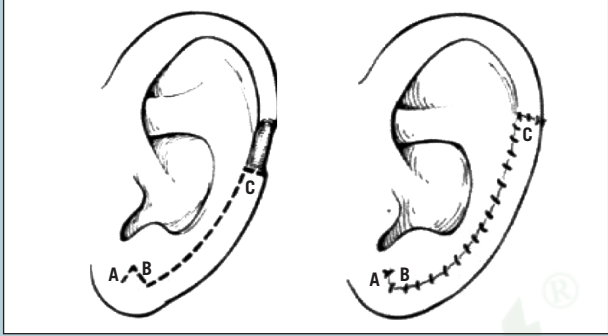
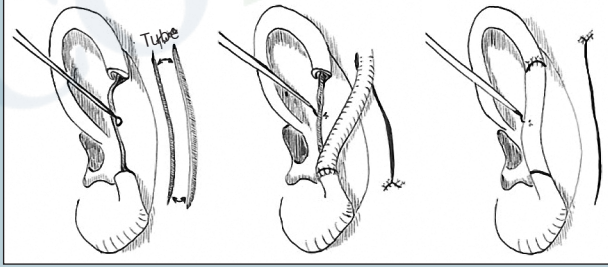
(Nose reconstruction algorithm cont)

Middle third	Central, lateral	Horizontal, round	Miter flap
		Vertical	V to Y flap
	Combined		Forehead flap
Distal third	Alar	Nasolabial flap, V to Y flap	
	Domal-alar groove		
	Dome	Bilobed flap	
	Central tip		
	Columella	Composite graft, FTSG	
	Nasal sill	Nasolabial flap	
	Combined	Forehead flap, nasolabial flap	
Combined	Forehead flap		

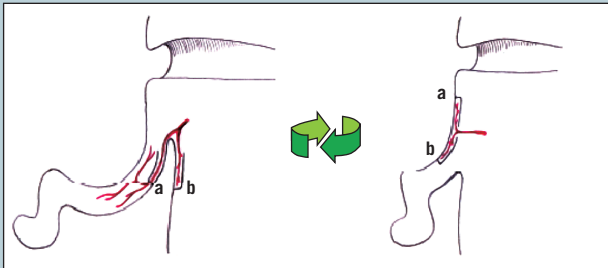
Ear Reconstruction

- Cartilage is only present in the upper two-thirds of the ear
- Superficial defects (except lobule and helical rim) can be managed with
 - Healing by secondary intention; presence of underlying cartilage prevents distortion from wound contraction
 - FTSG, only if perichondrium is available as recipient site
- Reconstruction is based on the anatomical unit, size, and layer involvement (skin versus composite)

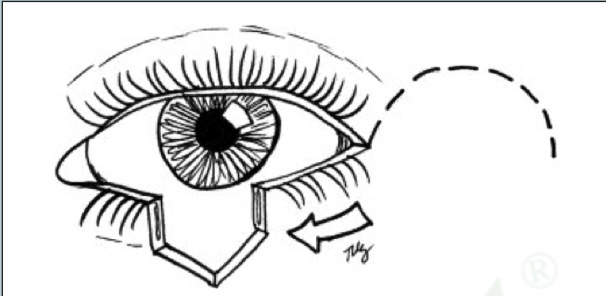
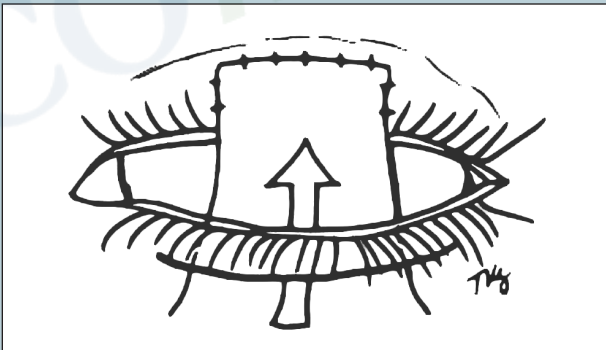
Ear reconstruction options

	Size	Flap choice
Helical rim	< 15% loss	<ul style="list-style-type: none"> • Helical advancement flap (Fig 8-12) • Wedge excision • Star excision; when wedge excision results in “cupping” deformity  <p>Fig 8-12 Helical advancement flap. (Modified with permission from Hayes CM. The ear: Excision and repair. Dermatol Clin 1998;16:109–125.)</p>
	> 15% loss	<p>Tubed bipediced postauricular flap (Fig 8-13); three-stage procedure</p>  <p>Fig 8-13 Tubed bipediced postauricular flap. (Reprinted with permission from Shonka DC, Park SS. Ear defects. Facial Plast Surg Clin North Am 2009;17:429–443.)</p>
Lobule	Small defect	Primary closure
	Large defect (> 30%)	<p>Local cutaneous flap</p> <p>Anterior-based auriculomastoid flap</p>

(Ear reconstruction options cont)

Conchal bowl	Superficial defect	<ul style="list-style-type: none"> • Primary closure • Secondary intention
	Composite defect	<p>Revolving door island flap (Fig 8-14); postauricular flap based on posterior auricular artery</p>  <p>Fig 8-14 Revolving door flap. (Reprinted with permission from Ruiz M, Garcia O, Hernán I, Sancho J., Serracanta J, Barret JP. Revolving-door flap: An alternative for the coverage of acute burn defects of the auricle. <i>Burns</i> 2011;37:e41–e43.)</p>
Upper ⅓ of the ear	<ul style="list-style-type: none"> • Composite defect requires both cartilage and skin reconstruction • Cartilage can be harvested from septum, contralateral ear, or rib • Reconstruction can be done with <ul style="list-style-type: none"> – Mladik pocket technique <ul style="list-style-type: none"> ◦ Graft is stored in a pocket that is created in postauricular skin ◦ Cartilage elevation 6–8 weeks later ◦ STSG is placed during the second-stage procedure for creation of a postauricular sulcus – Temporoparietal fascia flap <ul style="list-style-type: none"> ◦ Single stage procedure ◦ Temporoparietal fascia based on superficial temporal artery; fascia helps to provide blood supply to the underlying cartilage and the covering STSG 	
Lower ⅔ of the ear	<ul style="list-style-type: none"> • Composite defect requires both cartilage and skin reconstruction • Reconstruction is done using – <ul style="list-style-type: none"> Postauricular skin/cervical skin flap <ul style="list-style-type: none"> ◦ Requires two stages ◦ Cartilage is buried under this flap for 4 weeks ◦ STSG is used to create postauricular sulcus 	
Complete auricular defects	<ul style="list-style-type: none"> • Microvascular anastomosis if defect is related to traumatic avulsion <ul style="list-style-type: none"> – No venous anastomosis available, may require leech therapy postoperatively • Pocket technique—multiple stages (Brent technique) <ul style="list-style-type: none"> – First stage: Cartilage harvested from rib (sixth to ninth) and carved into an auricular shape; cartilage is stored in postauricular skin pocket for 3 months – Second stage: Lobule reconstruction – Third stage: Auricle graft is elevated – Final stage: Tragus reconstruction • Synthetic implant +/- craniofacial implants 	

Eyelid Reconstruction

	Size	Management
Upper eyelid	Small defect (< 30%)	Direct closure +/- lateral cantholysis
	Moderate defect (30% to 60%)	<ul style="list-style-type: none"> • Tenzel flap (Fig 8-15) • Sliding transconjunctival flap + FTSG  <p>Fig 8-15 Tenzel flap. (Reprinted with permission from Renner G, Kang T. Periorbital reconstruction: Brows and eyelids. Facial Plast Surg Clin North Am 2005;13:253-265.)</p>
	Large defect (> 60%)	Cutler-Beard flap (Fig 8-16); pedicle flap from lower lid (similar to Abbé flap in lip reconstruction)  <p>Fig 8-16 Cutler-Beard lid-sharing flap. (Reprinted with permission from Renner G, Kang T. Periorbital reconstruction: Brows and eyelids. Facial Plast Surg Clin North Am 2005;13:253-265.)</p>
Lower eyelid	Small defect	<ul style="list-style-type: none"> • Direct closure <ul style="list-style-type: none"> – 30% defect in young patient – 45% defect in older patient • Lateral cantholysis; can provide additional 5 mm of tissue advancement
	Moderate defect (< 60%)	Tenzel flap
	Large defect (> 60%)	<ul style="list-style-type: none"> • Hughes procedure: Two-stage procedure (Fig 8-17) • Mustardé rotational cheek flap

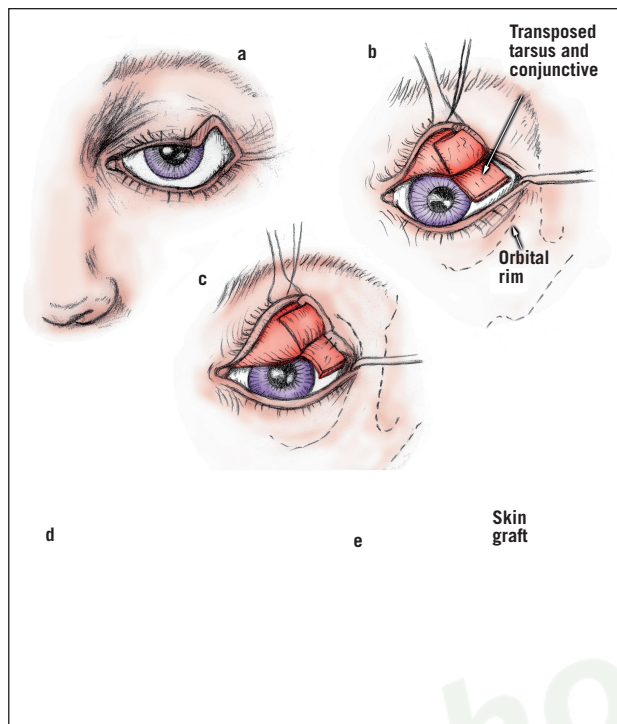


Fig 8-17 (a to e) Hughes procedure. (Reprinted with permission from Bashour M. Upper Eyelid Reconstruction Procedures Treatment & Management. Treatment: Surgical Therapy. Medscape. www.emedicine.medscape.com/article/1282054-treatment. Accessed 29 April 2015.)

Cheek Reconstruction

- Treatment should be based on skin laxity
- Hide incision along edge of nose, nasolabial fold, infraorbital rim, and preauricular crease

Size/type	Management
Small defect (< 2 cm)	Primary closure
Moderate defect (2–6 cm)	Local flap: Rhomboid flap, advancement flap, bilobed flap, rotational-advancement flap (Mustardé flap)
Large defect	Cervicofacial rotation flap
Through and through defect (without osseous component)	<ul style="list-style-type: none">• Chimeric free flap: ALT flap, thoracodorsal arterial flap, or parascapular flap• Single flap with portion of skin to be de-epithelialized
Through and through defect (with osseous component)	<ul style="list-style-type: none">• Double free flap: Fibular and ALT flaps• Single fibular flap with large skin paddle (de-epithelialize a portion of the skin paddle)

Lip Reconstruction

Reconstruction goals

- Oral sphincter competence
- Sensation
- Maintenance of stomal diameter

Vermilion reconstruction

Small defect	<ul style="list-style-type: none"> • Primary closure • V to Y advancement
Large defect	<ul style="list-style-type: none"> • Switch flap; requires second-stage surgery • Sliding advancement flap • Facial artery musculomucosal flap • Tongue flap; based on the ventral and lateral surface of the tongue

Lower lip reconstruction

> ½ defect	Primary closure
⅓ to ⅔ defect	<ul style="list-style-type: none"> • Reverse Abbé flap • Schuchardt procedure • Johansson step procedure
Commisure involvement	Estlander flap
> ⅔ defect (subtotal)	<ul style="list-style-type: none"> • Double reverse Abbé flap • Gilles flap • Karapandzic flap • Modified Webster-Bernard flap
Total defect	<ul style="list-style-type: none"> • Gate flap • Free radial-forearm flap with palmaris longus tendon

Upper lip reconstruction

Classified based on location and size: Central, lateral, and subtotal.

Central unit (philtrum) defect	<ul style="list-style-type: none"> • Primary closure: < one subunit • Abbé flap: Entire segment
Lateral unit	<ul style="list-style-type: none"> • Primary closure: < 50% defect • Abbé flap: Entire segment • Estlander flap, if commissure involved
Subtotal and total	Free radial-forearm flap

Midface Reconstruction

- Bone grafting can be done together with soft tissue flap
- Craniofacial prosthesis can provide both excellent functional and esthetic outcomes
- Reconstruction should be based on
 - Volume and size of soft tissue defect
 - Palatal closure
 - Orbital floor involvement
 - Need of orbital exenteration
- Classified based on the extent of maxilla that is resected and orbital and nasal involvement
 - JS Brown’s classification: Provides information on both vertical and horizontal dimensions (Fig 8-18)

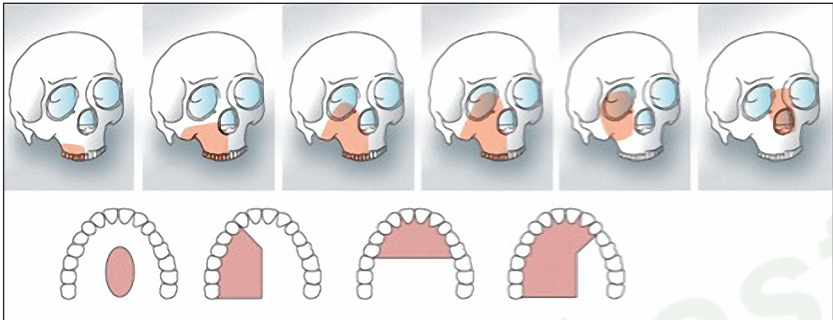


Fig 8-18 James Brown midface defect classification. (Reprinted with permission from Brown JS, Shaw RJ. Reconstruction of the maxilla and midface: Introducing a new classification. *Lancet Oncol* 2010;11:1001–1008.)

Vertical component (I-VI)	Horizontal component (A-D)
Type I: Maxillectomy with no oroantral fistula	Type A: Palatal defect only
Type II: Low maxillectomy (not involving orbit)	Type B: ≤ ½ unilateral palate
Type III: High maxillectomy (involving the orbital adnexae and orbital content)	Type C: Palatal defect crosses midline
Type IV: Radical maxillectomy (requires orbital exenteration)	Type D: > ½ maxillectomy
Type V: Isolated midface without alveolar component (orbitomaxillary defect)	
Type VI: Central component (nasomaxillary defect)	

- Moderate defect (type I and II): Prosthesis is as effective as free flap reconstruction
- Extensive defect: May require both prosthesis and free flap reconstruction to achieve optimal results
- Soft tissue coverage useful to provide barriers from cerebrospinal fluid leak and oronasal communication

Reconstruction options

	Maxillary moderate defect (types I and II)	Maxillary severe defect (types III and IV)	Nonmaxillary midface defect (types V and VI)
Soft tissue flap	Free radial-forearm flap and/or ALT flap	ALT flap, rectus flap	Free radial-forearm flap
Composite flap	Fibular flap	<ul style="list-style-type: none"> • Deep circumflex iliac arterial flap • Scapular flap • Thoracodorsal angular arterial flap with scapular tip 	Thoracodorsal angular arterial flap with scapular tip

Mandibular Reconstruction

Soft tissue	Technique	Type
Adequate	Nonvascularized technique <ul style="list-style-type: none"> • Lateral defect • < 9 cm in length 	<ul style="list-style-type: none"> • Iliac bone graft • Rib graft if condyle is involved • Bone morphogenetic protein
	Vascularized technique <ul style="list-style-type: none"> • Anterior mandibular defect • > 9 cm in length 	<ul style="list-style-type: none"> • Free fibular flap • Deep circumflex iliac arterial flap
Inadequate	Nonvascularized technique	Two-step approach <ul style="list-style-type: none"> • First stage: Soft tissue reconstruction (pectoralis major flap) • Second stage: 6 months later, iliac bone graft
	Vascularized technique	<ul style="list-style-type: none"> • Free fibular flap • Parascapular flap

Autogenous Bone Grafting

- Provides both osteoconductive and osteoinductive properties
- Requires vascularized soft tissue coverage at the recipient site and watertight closure
- Risk for failure
 - Anterior mandible
 - Defect > 9 cm
 - Inadequate soft tissue closure
 - Prior radiation
- Amount of bone needed: 10 cc of uncompressed bone per 1 cm of defect (based on panoramic radiograph)

Regional Bone Graft

	Cortical block size	Cancellous volume	Advantages	Disadvantages
Ramus	3 × 3 cm	2.3 cc	<ul style="list-style-type: none"> • Ease of harvesting • Intraoral donor site 	<ul style="list-style-type: none"> • Minimal cancellous bone • Risk of inferior alveolar nerve injury
Symphysis	2 × 1 cm	4.7 cc	<ul style="list-style-type: none"> • Ease of harvesting • Intraoral donor site 	Anterior mandibular teeth paresthesia

Distant Bone Graft

	Cortical block size	Cancellous volume	Advantages	Disadvantages
Calvarium	Abundant	Minimal	Abundant cortical bone	<ul style="list-style-type: none"> • Difficult to harvest • Minimal cancellous bone available • Difficult to do in outpatient setting
Anterior iliac	4 × 5 cm	< 50 cc	<ul style="list-style-type: none"> • Ease of harvesting • Abundant cancellous bone • Outpatient procedure is possible (trephine technique) 	<ul style="list-style-type: none"> • Risk of paresthesia at lateral thigh • Gait disturbance immediately postoperatively
Posterior iliac	5 × 5 cm	< 100 cc	Large amount of cancellous bone	<ul style="list-style-type: none"> • Requires positional change • Gait disturbance
Tibia	1 × 2 cm	< 25 cc	<ul style="list-style-type: none"> • Limited morbidity • Outpatient procedure is possible (trephine technique) 	Minimal cortical bone

Anterior Iliac Bone Graft (AIBG)

Surface anatomy	<ul style="list-style-type: none"> • ASIS • Tubercle of the ilium (6 cm posterior to ASIS)
Nerves	<ul style="list-style-type: none"> • Iliohypogastric (L1, L2) <ul style="list-style-type: none"> – Runs over the tubercle of the ilium – Most commonly affected nerve • Subcostal (T12, L1) <ul style="list-style-type: none"> – Runs over the tip of the anterior superior spine and slightly inferior to the iliohypogastric nerve • Lateral femoral cutaneous nerve <ul style="list-style-type: none"> – Courses within 1 cm of ASIS inferiorly
Muscles	<ul style="list-style-type: none"> • Medial aspect of ilium <ul style="list-style-type: none"> – External oblique muscle – Transverse abdominal muscle – Iliacus muscle • Lateral aspect of ilium <ul style="list-style-type: none"> – Tensor fascia lata; most important structure related to gait disturbance – Gluteus medius and minimus • Structures attached to ASIS <ul style="list-style-type: none"> – Inguinal ligament – Sartorius
Incision placement	<p>Incision (4–6 cm)</p> <ul style="list-style-type: none"> • Lateral to the anterior iliac spine • Placed between tubercle of the ilium and 1 cm proximal to the ASIS
Surgical technique	<p>Surgical layers</p> <ul style="list-style-type: none"> • Skin • Subcutaneous tissue • Scarpa fascia • Fibrous periosteum: Fuses between the tensor fascia lata laterally and external abdominal muscles medially <p>Exposure/harvesting of the bone</p> <ul style="list-style-type: none"> • Reflection of the iliacus muscle to expose the medial aspect of the ilium • Bone harvesting <ul style="list-style-type: none"> – Anteroposteriorly: 4–6 cm, preservation of 1 cm of bone posterior to ASIS to limit the risk of fracture/gait disturbance – Depth: 5 cm, where the medial and lateral cortex begins to fuse
Modification	<p>Different bone harvesting methods</p> <ul style="list-style-type: none"> • Clamshell or trapdoor approach (Figs 8-19a and 8-19b): Expand the medial and lateral cortices to gain access to the underlying cancellous bone • Tschopp approach (Fig 8-19c): Gain access to the cancellous bone by lateralizing the iliac crest (remains attached to the lateral muscle group) • Tessier approach (Fig 8-19d): Gain access to the cancellous bone by creating oblique osteotomies in the medial and lateral iliac crest (remains attached to the related muscle group)

ASIS, anterior superior iliac spine.

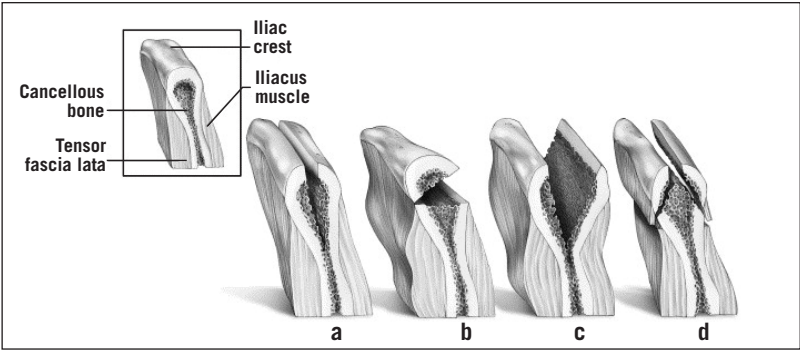


Fig 8-19 Modified AIBG harvesting methods. (a) Clamshell approach. (b) Trapdoor approach. (c) Tschopp approach. (d) Tessier approach. (Reprinted with permission from Kademani D, Keller E. Iliac crest grafting for mandibular reconstruction. *Atlas Oral Maxillofac Surg Clin North Am* 2006;14:161–170.)

Posterior Iliac Bone Graft (PIBG)

Surface anatomy	<ul style="list-style-type: none"> • Midline • Posterior iliac crest • Triangular fossa
Nerves	Sensory nerves <ul style="list-style-type: none"> • Superior cluneal nerve (L1–L3) • Middle cluneal nerve (S1–S3) Motor nerve <ul style="list-style-type: none"> • Sciatic nerve <ul style="list-style-type: none"> – 6–8 cm inferior to the posterior iliac crest – Should not be encountered during dissection
Muscles	<ul style="list-style-type: none"> • Gluteus maximus • Gluteus medius
Incision placement	<ul style="list-style-type: none"> • 6- to 8-cm curvilinear incision over posterior iliac crest • Incision should be placed between <ul style="list-style-type: none"> – Superior extension—insertion of the gluteus maximus – Inferior extension—paramedian and 3 cm lateral to the gluteal crease
Surgical technique	Positioning <ul style="list-style-type: none"> • Prone position • 210-degree jackknife position • Hip roll is placed to help define bony landmarks Surgical layers <ul style="list-style-type: none"> • Skin • Subcutaneous tissue • Thorcodorsal fascia • Periosteum Exposure/harvesing of the bone <ul style="list-style-type: none"> • Reflection of the gluteus maximus and medius • Bone harvesting <ul style="list-style-type: none"> – 5 × 5-cm cortical bone – < 100 cc uncompressed bone

Comparison of AIBGs and PIBGs

	AIBG	PIBG
Mean first day of ambulation	3.6 days	1.7 days
Seroma	Less (1%)	More (6%)
Hematoma	More (9%)	Less (4%)
Infection	More (1%)	Less (0%)
Pain	More	Less
Hyperesthesia	More (1%)	Less
Gait disturbance	More (32% after 2 weeks)	Less (6% after 2 weeks)
Mean operating time	Less	More
Other complications	<ul style="list-style-type: none"> • Injury to lateral femoral cutaneous nerve (maralgia paraesthetica) • Ileus • Abdominal hernia • Bleeding from gluteal artery 	<ul style="list-style-type: none"> • Ureteral injury • Paresthesia • Bleeding from subgluteal artery

Tibial Bone Graft

Surface anatomy	<ul style="list-style-type: none"> • Tibial plateau • Gerdy tubercle • Tibial midline
Nerve	Cutaneous branches of the lateral sural nerve
Muscle	<ul style="list-style-type: none"> • Anterior tibialis muscles (applies to lateral approach only); inferior to Gerdy tubercle • Pes anserinus (applies to medial approach only); attachment to the <ul style="list-style-type: none"> – Sartorius – Gracilius – Semitendinosus
Arteries	<ul style="list-style-type: none"> • Inferior genicular artery • Recurrent anterior tibial artery

Surgical techniques	<p>Skin layers</p> <ul style="list-style-type: none"> • Skin • Subcutaneous tissue • Iliotibial tract • Periosteum <p>Lateral approach</p> <ul style="list-style-type: none"> • Skin marking <ul style="list-style-type: none"> – Tibial head – Gerdy tubercle • Incision <ul style="list-style-type: none"> – 3- to 4-cm incision is made directly over Gerdy tubercle – Dissection down to the bone – 2-cm cortical window is made with fissure bur – Harvest cancellous bone through the cortical window (< 25 cc) across the plateau and down the shaft – Harvesting bone superiorly should be discouraged in case of knee joint violation • Closing <ul style="list-style-type: none"> – Multiple layer – No drain is needed <p>Medial approach</p> <ul style="list-style-type: none"> • Skin marking <ul style="list-style-type: none"> – Tibial head (tibial parallel line) – Tibial perpendicular line (midline of the shaft) • Incision <ul style="list-style-type: none"> – 15 mm inferior to the tibial perpendicular line and 15 mm medial to the tibial parallel line – Rest of the dissection and harvesting is similar to the lateral approach • Advantages <ul style="list-style-type: none"> – Encounters less vital structures (artery) – Further away from the knee joint – Similar amount of bone and complication rate – Stays within anterior compartment • Disadvantage <ul style="list-style-type: none"> – Skin marking is less objective, especially in obese patients
Complications (1% to 5.5%)	<ul style="list-style-type: none"> • Gait disturbance; relatively minor compared to iliac bone graft • Violation of joint space • Prolonged pain • Hematoma/seroma • Compartment syndrome (lateral approach only)

Costochondral Graft

Surface anatomy	<ul style="list-style-type: none"> • Inframammary crease • Sternum • Midaxillary line • Outline of the desired rib
Neurovascular	<ul style="list-style-type: none"> • Intercostal neurovascular bundle <ul style="list-style-type: none"> – Inferior border of the rib – Vein, artery, nerve (superior to inferior)
Rib selection	<p>Temporomandibular joint (TMJ) reconstruction</p> <ul style="list-style-type: none"> • Contralateral rib for better contouring • Use fifth to seventh ribs • If two ribs are needed <ul style="list-style-type: none"> – Avoid bilateral harvesting to prevent atelectasis – Use every other rib <p>Nasal reconstruction</p> <ul style="list-style-type: none"> • Use ninth to eleventh ribs <p>Ear reconstruction</p> <ul style="list-style-type: none"> • Use fifth to eighth rib
Surgical technique	<p>Incision</p> <ul style="list-style-type: none"> • Inframammary crease • From midaxillary line to sternum <p>Surgical layers</p> <ul style="list-style-type: none"> • Skin • Subcutaneous tissue • Pectoralis or rectus abdominis muscle • Fascia • Periosteum <p>Surgical tips</p> <ul style="list-style-type: none"> • For TMJ reconstruction <ul style="list-style-type: none"> – 4 to 10 mm of cartilage is needed; too much cartilage = overgrowth and asymmetry – 3 to 4 cm of bone – 1 mm of periosteum and perichondrium intact at the costochondral junction to minimize the risk of cartilage separation

Complications	<p>Pneumothorax or pleural laceration</p> <ul style="list-style-type: none">• 4% to 30%• Inspection: Inflate the lung while rinsing the harvest site with saline; presence of bubbles may indicate pleural laceration• Management<ul style="list-style-type: none">– Small tear: Suture pleura over a red rubber catheter– Large tear: Chest tube insertion– Chest radiograph should be obtained postoperatively to evaluate for residual pneumothorax <p>Atelectasis</p> <ul style="list-style-type: none">• Caused by postoperative pain and poor respiratory effort• Management<ul style="list-style-type: none">– Intercostal nerve block before wound closure– Incentive spirometry <p>Thoracic scoliosis</p> <ul style="list-style-type: none">• Incidence rate as high as 25%• Risk<ul style="list-style-type: none">– Microtia repair– Age < 10 years old <p>Chronic pleuritic chest pain</p>
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Recommended Readings

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Orofacial Pain

David W. Lui and Daniel M. Laskin

- ▶ Neuroanatomy and Neurophysiology of Orofacial Pain
- ▶ Neuropathic Pain Disorders
- ▶ Headache
- ▶ Masticatory Muscle Disorders

Neuroanatomy and Neurophysiology of Orofacial Pain

- Three major branches (V1–V3)
- Three major nuclei
 - Bilateral columns in brainstem (midbrain to dorsal horn of cervical spine)
 - Rostral-caudal: Mesencephalic, main sensory, spinal trigeminal

	Foramen	Function
V1 (ophthalmic nerve)	Superior orbital fissure	<ul style="list-style-type: none">• Sensory only to upper third of the face
V2 (maxillary)	Foramen rotundum	<ul style="list-style-type: none">• Sensory to middle third of the face• Parasympathetic innervation from pterygopalatine ganglion to lacrimal gland
V3 (mandibular)	Foramen ovale	<ul style="list-style-type: none">• Sensory – Lower third of the face – Auriculotemporal nerve innervates most of temporo-mandibular joint• Motor innervation<ul style="list-style-type: none">– Muscles of mastication– Tensor veli palatinei– Tensor tympani

Trigeminal Nuclei

	Comments
Mesencephalic nucleus	<ul style="list-style-type: none">• Proprioception• Apical periodontal ligaments and jaw muscles (jaw-closing reflex)
Main sensory nucleus	<ul style="list-style-type: none">• Facial proprioception (except jaw-closing reflex)
Spinal trigeminal nucleus (medullary dorsal horn)	<p>Three subnuclei (SN)</p> <ul style="list-style-type: none">• Rostral-caudal: SN oralis, SN interpolaris, SN caudalis<ul style="list-style-type: none">– SN oralis and SN interpolaris<ul style="list-style-type: none">◦ Tactile◦ Temperature– SN caudalis<ul style="list-style-type: none">◦ Nociception

Common Orofacial Pain Terminology

	Comments
Acute pain versus chronic pain	<ul style="list-style-type: none"> • Acute pain: < 3 months • Chronic pain: ≥ 3 months <ul style="list-style-type: none"> – Reduced pain thresholds – Central behavioral changes – Refractory to acute pain therapy
Allodynia	Pain caused by a stimulus that does not normally cause pain
Analgesia	Absence of pain in response to normally painful stimuli
Anesthesia dolorosa	Pain in an area or region that is anesthetic
Dysesthesia	An unpleasant abnormal sensation, whether spontaneous or evoked
Heterotopic pain	Pain source is not located in the region of pain perception but is within the same dermatome
Hyperalgesia	An increased pain response (more pain) to a normally painful stimulus
Hypoalgesia	Diminished pain in response to a normally painful stimulus
Hypoesthesia	Decreased sensitivity to sensory stimulation (excludes the special senses)
Paresthesia	An abnormal sensation, whether spontaneous or evoked
Referred pain	Pain felt at a location innervated by one nerve, but the nociceptive impulses arrive on a different nerve (can be from a different dermatome)
Sensitization	<ul style="list-style-type: none"> • An increase in the excitability of neurons • Two mechanisms <ul style="list-style-type: none"> – Peripheral <ul style="list-style-type: none"> ◦ A reduction in threshold and an increase in responsiveness of the peripheral ends of nociceptors ◦ Example: Pain after sunburn ◦ Sensitization arises due to the action of inflammatory chemicals – Central <ul style="list-style-type: none"> ◦ Increase in the excitability of neurons within the central nervous system so that normal inputs begin to produce abnormal responses ◦ Example: Tactile allodynia

Neuropathic Pain Disorders

- Characteristics of episodic versus continuous neuropathic pain
 - Episodic: Severe, paroxysmal electric shock–like and/or lancinating pain of short duration
 - Continuous: Aching, burning, throbbing, stabbing pain
- Quantitative sensory testing
 - Heat activates C-fibers (unmyelinated, small-diameter, slow conduction)
 - Cold stimuli and punctate mechanical stimuli activate A-delta fibers (myelinated, large-diameter, fast conduction—afferent fibers only)
 - Electrical stimuli activate A-beta fibers (myelinated, large-diameter, fast conduction—both afferent and efferent fibers)

Episodic neuropathic pain	Continuous neuropathic pain
<ul style="list-style-type: none"> • Trigeminal neuralgia • Glossopharyngeal neuralgia 	<ul style="list-style-type: none"> • Idiopathic trigeminal neuropathic pain • Post-herpetic neuralgia • Central post-stroke pain • Complex regional pain syndrome (CRPS)

Episodic Neuropathic Pain Disorders

Trigeminal neuralgia (TN)	<ul style="list-style-type: none"> • Pathophysiology <ul style="list-style-type: none"> – Most common hypothesis: Vascular compression of trigeminal nerve root entry zone → demyelination → spontaneous nerve firing • Signs and symptoms: Excruciating, short-lasting, shock-like facial pain <ul style="list-style-type: none"> – Unilateral, R > L – V2 and V3 > V1 – No neurologic deficits – Frequently pain free between attacks – Local anesthesia of trigger point temporarily arrests pain • Trigger points: Areas in distribution of the affected trigeminal branch (lip, nose, eye, inside mouth) that initiate pain when touched • Types – Classic TN: 85% of cases – Atypical TN: Longer attacks and constant background pain – Symptomatic TN: Various forms of etiologic pathology including tumors, vascular malformations • Differential diagnosis <ul style="list-style-type: none"> – Multiple sclerosis – Intracranial tumor: Posterior and middle fossa
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(Episodic Neuropathic Pain Disorders cont)

	<ul style="list-style-type: none">• Treatment – Pharmacology<ul style="list-style-type: none">◦ 1st line: Carbamazepine◦ 2nd line: Phenytoin– Surgery<ul style="list-style-type: none">◦ Peripheral procedure<ul style="list-style-type: none">– Neurectomy –Cryotherapy –Alcohol injection◦ Central procedure<ul style="list-style-type: none">– Percutaneous rhizotomy– Microvascular decompression– Gamma Knife radiosurgery
Glossopharyngeal neuralgia (GN)	<ul style="list-style-type: none">• Pharyngeal GN<ul style="list-style-type: none">– Pain in pharynx, tonsil, soft palate, posterior tongue base– Pain radiates toward mandibular angle• Tympanic GN<ul style="list-style-type: none">– Pain in ear– Pain may radiate to pharynx (can be confused with geniculate neuralgia)• Pain triggered by swallowing, chewing, talking, coughing, yawning• Can induce bradycardia and syncope• Order magnetic resonance image (MRI) to rule out pathology (symptomatic GN)<ul style="list-style-type: none">– Tonsillar carcinoma in parapharyngeal space, cerebellopontine angle mass, vascular anomalies• Medical treatment similar to TN; intracranial cranial nerve IX and X upper rootlet sectioning, microvascular decompression

Continuous Neuropathic Pain Disorders

Idiopathic trigeminal neuropathic pain	<ul style="list-style-type: none">• Chronic dental or facial pain that does not meet any diagnostic criteria and does not respond to most treatments, females > males, maxilla > mandible• Symptoms<ul style="list-style-type: none">– Dull aching or throbbing pain, paresthesia– Present continuously for months or years with intermittent periods of increased pain• Subtypes<ul style="list-style-type: none">– Atypical odontalgia/phantom tooth pain: Pain confined to teeth and gingival area– Atypical facial pain: Pain involves parts of the face• Treatment: Tricyclic antidepressants, gabapentin, pregabalin, tramadol, topical lidocaine
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(Continuous Neuropathic Pain Disorders cont)

Post-herpetic neuralgia	<ul style="list-style-type: none">• Reactivation of latent infection by varicella zoster virus dormant in peripheral ganglion• 16% to 22% of patients experience neuralgia—burning, throbbing, shooting, or sharp pain; may be accompanied by allodynia, hyperalgesia, anesthesia dolorosa• Associated with pale or reddish scars that are hypoesthetic or anesthetic<ul style="list-style-type: none">– V1 distribution (80%) if cranial nerve V affected– Ramsay Hunt syndrome: Herpes zoster infection of sensory and motor branches of cranial nerve VII +/- cranial nerve VIII<ul style="list-style-type: none">◦ Associated with facial paralysis, vertigo, deafness, anesthesia dolorosa• Treatment: Tricyclic antidepressants, gabapentin, pregabalin, opioids, tramadol, topical lidocaine
Central post-stroke pain	<ul style="list-style-type: none">• Develops in approximately 8% of post-stroke patients• After a stroke, the brain does not understand normal messages sent from the body in response to touch, warmth, cold, and other stimuli• Pain, dysesthesia, and impaired sensation to pinprick and temperature stimulation; slight sensation may be painful• Pain may be constant or episodic; onset may be more than a month after stroke• Treatment<ul style="list-style-type: none">– 1st line: Amitriptyline, lamotrigine– 2nd line: Mexiletine, phenytoin– Intravenous ketamine, propofol, lidocaine
CRPS	<ul style="list-style-type: none">• Severe pain, swelling, and skin changes associated with history of nerve and tissue injury; sympathetic symptoms (ie, lacrimation)• Worsens over time• Associated with dysregulation of central nervous system and autonomic nervous system<ul style="list-style-type: none">– Sympathetic nervous system: Edema, cutaneous blood flow changes, motor changes, trophic changes– Often termed <i>hot</i> or <i>cold</i> CRPS based on skin temperature of affected area<ul style="list-style-type: none">◦ Hot CRPS—acute condition◦ Cold CRPS—chronic condition, poorer prognosis• Two types<ul style="list-style-type: none">– Type I (reflex sympathetic dystrophy)<ul style="list-style-type: none">◦ Presence of a noxious initiating event without nerve damage◦ Aching, pricking, burning pain– Type II (causalgia)<ul style="list-style-type: none">◦ Presence of a noxious initiating event with nerve damage◦ Electric and/or shooting pain• Treatment<ul style="list-style-type: none">– Physical therapy and/or occupational therapy, tricyclic antidepressants, selective serotonin reuptake inhibitors, anticonvulsants, gabapentin, steroids, opioids– Local anesthetic injections– Neurostimulation– Sympathectomy– Awake and/or subanesthetic low-dose intravenous ketamine infusion

Headache

Primary headaches	Secondary headaches
<ul style="list-style-type: none">• Migraine• Tension-type headache• Cluster headache• Paroxysmal hemicrania	<ul style="list-style-type: none">• Temporal arteritis• Posttraumatic headache

Primary Headache Disorders

- Primary versus secondary headache
 - Primary headache: No other cause
 - Secondary headache: Occurs in close temporal relation to another disorder

Migraine headache	<ul style="list-style-type: none">• Commonly starts at age 15–24, but most frequent at ages 35–45; females > males• Four possible phases: Prodromal, aura, pain, postdromal• Pathophysiology: Neurovascular disorder• Symptoms: Unilateral pulsing headache lasting 2–72 hours; associated with nausea, vomiting, and sensitivity to light, sound, and smell• Types<ul style="list-style-type: none">– Common migraine without aura– Classic migraine with aura– Chronic migraine: 15 days per month– Status migranosus: Lasts > 3 days• Treatment<ul style="list-style-type: none">– Symptomatic<ul style="list-style-type: none">◦ Specific: 5-hydroxytryptamine (5-HT) agonists (triptans), dihydroergotamine◦ Nonspecific: Acetaminophen, acetylsalicylic acid (ASA), nonsteroidal anti-inflammatory drugs (NSAIDs), caffeine– Prophylactic<ul style="list-style-type: none">◦ Most effective: Tricyclic antidepressants, sodium valproate, topiramate, timodol◦ Moderately effective: Gabapentin, verapamil, naproxen
Tension-type headache	<ul style="list-style-type: none">• Onset: Age 20–40• 23% of female population; 18% of male population• Pathophysiology<ul style="list-style-type: none">– Triggers: Stress, hunger, sleep deprivation, bad posture, eye strain– Classic theory: Muscle contraction/tension• Symptoms: Bilateral pressing/tightening (nonpulsatile) quality• Treatment<ul style="list-style-type: none">– Episodic type: NSAIDs, acetaminophen, ASA; frequent use of these drugs can lead to rebound headache– Chronic type: Amitriptyline, mirtazapine, relaxation techniques

(Primary Headache Disorders cont)

Cluster headache	<ul style="list-style-type: none">• Onset: 27–31 years• Males > females• Pathophysiology: Hypothalamic abnormality• Symptoms: Severe unilateral orbital, supraorbital, temporal pain lasting 15–180 minutes accompanied by ipsilateral lacrimation, rhinorrhea, eyelid edema, miosis; 1 to 8 attacks per day<ul style="list-style-type: none">– Symptomatic/abortive: Oxygen (7 L per minute for 15 minutes), triptans, dihydroergotamine– Prophylactic: Verapamil (1st line), short-term corticosteroid, lithium, divalproex sodium, topiramate– Intractable type: Invasive procedure (ie, sphenopalatine block, trigeminal rhizotomy, microvascular decompression of trigeminal nerve)
Paroxysmal hemicrania	<ul style="list-style-type: none">• Sjaastad syndrome• Similar to cluster headache, but<ul style="list-style-type: none">– Shorter duration: 2–30 minutes– More frequent– More common in females– 100% responsive to indomethacin

Secondary headaches

Temporal arteritis	<ul style="list-style-type: none">• Vasculitis of large and medium arteries of head, predominately external carotid artery branches (frequently temporal artery)• May rapidly lead to blindness due to giant-cell granulomatous occlusion of ophthalmic artery• Suspect if patient is > 50 years with new persistent headache centered on one or both temples, worsened by cold temperatures, associated with jaw claudication• Diagnosis<ul style="list-style-type: none">– Prominent temporal artery, temporal tenderness, decreased temporal pulse, ischemic fundus– Increased erythrocyte sedimentation rate and C-reactive protein –• Biopsy (gold standard): Unilateral temporal artery biopsy of 1.5 to 3 cm has 85% to 90% sensitivity• Treatment<ul style="list-style-type: none">– Corticosteroid as soon as possible before diagnosis confirmed by biopsy to prevent permanent blindness due to ophthalmic artery occlusion
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(Secondary headaches cont)

Posttraumatic headache	<ul style="list-style-type: none"> • Post-closed-head injury syndrome, postconcussion syndrome • Diagnostic criteria – Head trauma with persistent posttraumatic amnesia, loss of consciousness or posttraumatic seizure + neuropsychologic impairment + 3 of the following for > 3 months postinjury <ul style="list-style-type: none"> ◦ Headache, dizziness, fatigue, irritability, sleep problems, affect changes, personality changes, apathy • Treatment <ul style="list-style-type: none"> – Eliminate aggravating extracranial nociceptive pain sources – Neuropsychiatric disability rehabilitation – Tricyclic antidepressants, gabapentin, pregabalin
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Masticatory Muscle Disorders

- One component of the spectrum of temporomandibular disorders (TMDs) (see chapter 10)
- Intrinsic pain in masticatory muscles, sometimes associated with localized trigger points (expressed as taut bands)
- **Etiology**
 - Trauma: Direct macrotrauma, indirect macrotrauma (whiplash)
 - Parafunction: Bruxism, stress
- **Symptoms:** Pain, muscle tenderness, limited range of mandibular motion
- **Important clinical information**
 - Quality of pain: Dull, aching
 - Location: Diffuse versus localized; unilateral versus bilateral
 - Intensity: Mild to severe
 - Onset and duration: Episodic
 - Modifiers: Increases with function, stress
 - Comorbidities: Fibromyalgia, migraine headache, irritable bowel syndrome
- **Examination**
 - Palpation for muscle tenderness
 - Assessment of mandibular opening and function
 - Provocation tests
 - Trigger point stimulation
 - Pain on tooth clenching
 - Reproduction of symptoms with chewing

Subtypes of Masticatory Muscle Disorders

Myofascial pain	Repetitive strain: Unilateral, dull, aching pain; limited mouth opening; patients often complain of frequent headache and earache; soft end-feel; pain increases with function
Muscle spasm	Acute overuse: Characterized by acute, involuntary, and continuous muscle contraction; limited mouth opening; sensation of muscle cramping
Myositis	Injury or infection: Swelling and inflammation secondary to direct trauma or infection; trismus
Muscle contracture	Muscle fibrosis: Painless shortening of muscle; severe limitation of jaw function

Comparison of various masticatory muscle disorders

	Pain with function	Trismus	Comments
Myofascial pain	Yes	Mild to moderate	<ul style="list-style-type: none"> • Unilateral dull, aching pain • Localized, reproducible tenderness • Sensation of acute malocclusion (not verifiable clinically) • Otologic symptoms, complaints of frequent temporal headache
Muscle spasm	Yes	Moderate	<ul style="list-style-type: none"> • Acute onset of pain • Sensation of muscle tightness, cramping, stiffness
Myositis	Yes	Moderate	<ul style="list-style-type: none"> • Localized, continuous pain in muscle • Diffuse tenderness and swelling of the entire length of affected muscle
Muscle contracture	Usually	Severe	<ul style="list-style-type: none"> • Unyielding firmness on passive stretch (hard end-feel) • Little or no pain unless involved muscle is forced to lengthen • Remote history of trauma, infection, or long period of disuse and limited range of motion

Treatment Modalities

Multiple modalities are often needed. Always begin with conservative and reversible treatments.

Myofascial pain	<ul style="list-style-type: none">• Soft, nonchewy diet• Heat and massage• Splint therapy• Gentle range of motion exercises• NSAIDs for pain; avoid narcotics• Muscle relaxant drugs (cyclobenzaprine)• Tranquilizers (diazepam)• Soporific for sleep• Cognitive behavioral therapy
Muscle spasm	<ul style="list-style-type: none">• NSAIDs• Physical therapy<ul style="list-style-type: none">– Heat and massage– Mobilization– Electrogalvanic stimulation– Ultrasound– Transcutaneous electrical nerve stimulation (TENS)
Myositis	<ul style="list-style-type: none">• Due to Injury<ul style="list-style-type: none">– NSAIDs– Intermittent moist heat– Physical therapy when acute symptoms subside• Due to Infection<ul style="list-style-type: none">– NSAIDs– Antibiotics– Intermittent moist heat– Physical therapy when acute symptoms subside
Muscle contracture	<ul style="list-style-type: none">• Physical therapy• Surgery if physical therapy unsuccessful<ul style="list-style-type: none">– Muscle stripping– Myotomies

Fibromyalgia

- Characterized by muscle ache, joint stiffness, fatigue, sleep disturbance, multiple myofascial tender points (also in trunk and appendages)
- Mean age 52, 80% to 90% female
- Four subtypes
 - Extreme sensitivity to pain but not associated with psychiatric conditions; may respond to 5-HT₃ blocker
 - Fibromyalgia and comorbid, pain-related depression; may respond to antidepressants
 - Depression with concomitant fibromyalgia syndrome; may respond to antidepressants
 - Fibromyalgia due to somatization; may respond to psychotherapy
- **Pathophysiology:** Disruption of dopamine neurotransmission, dysregulated serotonin metabolism
- **Diagnostic criteria**
 - A history of widespread pain lasting > 3 months, affecting all four quadrants of the body (bilateral and above and below waist)
 - Tender points: There are 18 possible designated tender points (9 pairs); should have at least 11 of the 18 for a positive diagnosis
- **Treatment:** Pregabalin, duloxetine, milnacipran, psychologic counseling, cognitive behavioral therapy

Fibromyalgia versus myofascial pain

Not all myofascial pain patients have fibromyalgia, but all fibromyalgia patients have myofascial pain.

	Myofascial pain	Fibromyalgia
Pain distribution	Local or regional (face, head, neck, shoulders)	Diffuse pain involving all areas of the body
Tender areas	Tender points that produce pain distant to the location pressed	Tender points that are painful on local pressure
Fatigue	No	Yes
Sleep disturbance	Yes	Yes
Panic attacks	No	Yes
Depression	Sometimes	Always

Recommended Readings

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The TMJ

David W. Lui and Daniel M. Laskin

- ▶ Anatomy of the TMJ
- ▶ Surgical Anatomy of the TMJ
- ▶ TMJ Imaging
- ▶ Nonsurgical Management of TMJ Disorders
- ▶ Arthritis of the TMJ
- ▶ Internal Derangements of the TMJ
- ▶ Abnormal TMJ Growth Disorders
- ▶ TMJ Hypomobility and Hypermobility
- ▶ Neoplasms of the TMJ
- ▶ Alloplastic TMJ Reconstruction
- ▶ Complications in TMJ Surgery

Anatomy of the TMJ

Type of joint	<ul style="list-style-type: none">• Ginglymoarthrodial joint<ul style="list-style-type: none">– Superior compartment: Translational movement in arthrodial joint (sliding joint)– Inferior compartment: Rotational movement in ginglymus joint (hinging joint)• Four articulating surfaces<ul style="list-style-type: none">– Articular eminence of temporal bone– Superior surface of articular disc– Inferior surface of articular disc– Articular surface of mandibular condyle
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Components

Mandibular condyle	<ul style="list-style-type: none">• 145- to 160-degree horizontal angle of one condyle to the other• Dimensions<ul style="list-style-type: none">– Mediolateral 15 to 20 mm, with round surface– Anteroposterior 8 to 10 mm with convex surface• Formed by intramembranous process
Articular cartilage	<ul style="list-style-type: none">• Articular surface covered by connective tissue layer (perichondrium)• Underlying hyaline cartilage composed of<ul style="list-style-type: none">– Chondrocytes (arranged in three zones)<ul style="list-style-type: none">◦ Superficial: Small flat cells with long axis parallel to surface◦ Middle: Larger rounded cells in columnar pattern perpendicular to surface◦ Deep: Largest cells above “tide mark” (demarcation from calcified subchondral area)– Type II collagen – Water<ul style="list-style-type: none">◦ 65% to 80% of cartilage mass◦ Stress-shielding of solid matrix components due to incompressibility of water content– Ground substance: Glycosaminoglycans<ul style="list-style-type: none">◦ Polysaccharide side chains attached to the core proteins of the proteoglycans◦ Chondroitin sulfate: Evenly distributed in cartilage
Articular part of temporal bone	<ul style="list-style-type: none">• Articular fossa (glenoid fossa)<ul style="list-style-type: none">– Concave surface extending from posterior slope of articular eminence to postglenoid tubercle– Mean thickness of 0.9 mm• Articular eminence – Mediolateral bony prominence forming the posterior root of zygomatic arch and anterior wall of articular fossa<ul style="list-style-type: none">– Not to be confused with articular tubercle, which is located lateral to the articular eminence and is nonarticular• Preglenoid plane: Flattened area located anterior to the articular eminence

(Components cont)

Articular disc	<ul style="list-style-type: none"> • Nonvascularized, noninnervated except at the periphery • Three zones <ul style="list-style-type: none"> – Anterior band—thickest part – Intermediate zone—thinnest part – Posterior band • Attached to capsule, retrodiscal tissue and some fibers of superior head of lateral pterygoid muscle • Retrodiscal tissue (bilaminar zone) <ul style="list-style-type: none"> – Highly vascular and innervated – Produces synovial fluid – Superior retrodiscal lamina: Contains elastic fibers that prevent extreme translational movements and aid in repositioning the disc during mouth closure – Inferior retrodiscal lamina: Collagen fibers, prevents extreme rotational movements
Synovial cavity	<ul style="list-style-type: none"> • Divided into two spaces by the articular disc <ul style="list-style-type: none"> – Superior joint space—largest volume – Inferior joint space • Lined by synovial membrane (synovial intima cells); two types of synovial cells <ul style="list-style-type: none"> – Type A synovial cells <ul style="list-style-type: none"> ◦ Macrophage-like activity: Ingestion of debris via endocytosis ◦ Produces most of hyaluronic acid for lubrication ◦ Predominantly in fetal synovium – Type B synovial cells <ul style="list-style-type: none"> ◦ Produces proteoglycans and glycoproteins for lubrication of cartilaginous surfaces ◦ Fibroblast-like appearance ◦ Predominantly in older patients • Synovial fluid – Ultrafiltrate of plasma <ul style="list-style-type: none"> ◦ Lower total protein content ◦ Higher percentage of albumin ◦ Lower percentage of gamma globulin ◦ Alkaline phosphatase ◦ White blood cell count < 200 per cc (< 25% polymorphonuclear leukocytes) – Average volume < 2 cc – Hyaluronic acid for viscosity – Lubrication of joint, nourishment of articular cartilage

(Components cont)

Ligaments	<ul style="list-style-type: none"> • Functional ligaments <ul style="list-style-type: none"> – Collateral (discal) <ul style="list-style-type: none"> ◦ Attaches disc to lateral and medial poles of condyle ◦ Resists movement of disc away from condyle ◦ Permits rotation of disc with condyle and maintains condyle-disc relationship during translational movement – Capsular <ul style="list-style-type: none"> ◦ Extends from condylar neck to glenoid fossa and eminence ◦ Resists medial, lateral, and inferior forces ◦ Contains synovial fluid in joint – Temporomandibular <ul style="list-style-type: none"> ◦ Outer oblique part limits rotational movement ◦ Inner horizontal part limits posterior movement • Accessory ligaments <ul style="list-style-type: none"> – Sphenomandibular: Limits excessive mouth opening
Vascular supply	<ul style="list-style-type: none"> • Anterior: Superficial temporal and maxillary arteries • Posterior: Masseteric artery
Innervation	<ul style="list-style-type: none"> • Auriculotemporal nerve (main innervation) • Masseteric nerve • Posterior deep temporal nerve
Associated musculature	<ul style="list-style-type: none"> • Supramandibular group <ul style="list-style-type: none"> – Lateral pterygoid: Anterior translation in conjunction with digastric; opens the mouth; in conjunction with medial pterygoid; promotes side-to-side movement (grinding) – Medial pterygoid: Mouth closure, protrusion – Masseter: Mouth closure, protrusion – Temporalis (anterior head): Mouth closure, protrusion – Temporalis (posterior head): Mouth closure, retrusion • Inframandibular group <ul style="list-style-type: none"> – Suprahyoid <ul style="list-style-type: none"> ◦ Digastric, geniohyoid, mylohyoid, stylohyoid ◦ Raises hyoid bone if mandible stabilized by supramandibular group ◦ Depresses mandible if hyoid stabilized by infrahyoid group – Infrahyoid <ul style="list-style-type: none"> ◦ Sternohyoid, omohyoid, sternothyroid, thyrohyoid ◦ Depresses or stabilizes hyoid bone

Surgical Anatomy of the TMJ

Pertinent Surgical Anatomy

Facial nerve: Temporal branch

- Located beneath the superficial muscular aponeurotic system (SMAS) layer/temporoparietal fascia
- Mean distance between temporal branch and anterior border of bony external auditory canal: 0.8 to 3.5 cm (Al-Kayat study)
- Becomes superficial to the SMAS layer at
 - 1.5 to 3 cm above zygomatic arch
 - 1.5 cm lateral to orbital rim

Vascular structures

- Superficial temporal vessels: Superficial to the SMAS layer
- Internal maxillary artery
 - Relevant when condylectomy is performed
 - Medial to the condylar process
 - 20 mm below the condylar head

Facial Layers to Access TMJ Capsule

- Skin
- Subcutaneous tissue
- SMAS
- Superficial layer of deep temporalis fascia
- Periosteum of the zygomatic arch or lateral capsule of TMJ

Joint Space Entrance

To enter the superior joint space, a horizontal incision is made through the capsule just below the superior border of the glenoid fossa. The incision should be made from anterior to posterior to approach the retrodiscal tissue area last. To enter the inferior joint space, an incision is made below the point of attachment of the disc to the capsule parallel to the superior incision.

- Exposure of the condylar process
 - T incision is made over the lateral capsule
 - The horizontal incision is similar to the one used to enter the upper joint space
 - The vertical incision is made extending inferiorly in the middle of the condylar neck
 - Caution should be used to avoid any damage to the cartilage on the condylar head
 - Cutting the retrodiscal tissue can lead to excessive bleeding; control bleeding by reseating the condyle in the fossa and maintaining pressure for several minutes and/or by using electrocautery

Common Open Joint Incision Designs

Preauricular	<ul style="list-style-type: none">• Most common incision used in TMJ surgery• Incision is placed in preauricular crease• Superior border: Uppermost attachment of the auricle• Inferior border: Few millimeters below the lowermost attachment of the auricle• Modifications<ul style="list-style-type: none">– Endaural<ul style="list-style-type: none">◦ Skin incision wraps around the posterior aspect of the tragus◦ During closing, make sure to reapproximate the subcutaneous tissue to the base of the tragus to avoid tragal blunting– Al-Kayat<ul style="list-style-type: none">◦ Superior extension of the incision in temporal region◦ Helpful if temporalis flap is needed for grafting purposes
Postauricular	<ul style="list-style-type: none">• Not commonly used but most esthetic• Increases operating time• Incision is placed 3 to 4 mm posterior to the auricular flexure• The external auditory canal is completely transected (360 degrees) and the ear is retracted anteriorly to expose the joint• Can result in stenosis of the external auditory canal
Retromandibular	<ul style="list-style-type: none">• 3-cm vertical incision is made 1 cm posterior to the posterior border of the ramus; after undermining the skin to gain access, the tail of the parotid is retracted posteriorly exposing the underlying masseter, which is incised vertically to expose the mandible• Provides good access for open reduction of subcondylar fractures• Can be used to access the TMJ area in young children because of the relatively short ramus
Submandibular	<ul style="list-style-type: none">• Risdon incision• 4-cm long incision is made 1 cm below the most inferior point of mandibular angle• Usually combined with preauricular incision for total joint reconstruction<ul style="list-style-type: none">– 2-cm skin area should be left intact between these two incisions• Vital structures at risk<ul style="list-style-type: none">– Marginal mandibular branch of cranial nerve VII– Retromandibular vein

TMJ Imaging

Most commonly used imaging techniques for the TMJ

- Panoramic radiograph: Initial screening
- Computed tomography (CT) scan: Osseous TMJ pathology and fabrication of custom TMJ prostheses
- Magnetic resonance imaging (MRI): Evaluation of intra-articular soft tissue pathology (internal derangements)
- Scintigraphy: Shows bone building or rebuilding and acute and chronic inflammation

Signal Intensity Characteristics of MRI T1- and T2-Weighted Images

	Bright signal intensity	Low signal intensity
T1 weighted (short TR, short TE)	<ul style="list-style-type: none">• Fat: Fatty bone marrow• Blood (if subacute)	<ul style="list-style-type: none">• Fibrous tissue: Disc, tendons• Water: Edema, tumor, inflammation• Muscle, calcium, air
T2 weighted (long TR, long TE)	<ul style="list-style-type: none">• Water: Edema, tumor, inflammation• Blood (if subacute)	<ul style="list-style-type: none">• Fat, fibrous tissue• Paramagnetic substances (hemosiderin)
TE, echo time; TR, repetition time.		

Normal TMJ MRI Features

- Closed-mouth view
 - Posterior band of disc at the 11- to 12-o'clock position of condylar head
 - Disc is biconcave and has low MR signal intensity
- Open-mouth view
 - Thinnest intermediate zone interposed between condyle and articular eminence, disc morphology remains biconcave
- Condyle marrow MR signal intensity
 - T1: Bright
 - T2: Less bright

Scintigraphy

Technetium-99	<ul style="list-style-type: none">• Taken up by osteoclasts• Shows bone building or rebuilding
Gallium-67	<ul style="list-style-type: none">• Uptake by tumors, areas of acute or chronic inflammation• Good for diagnosis of osteomyelitis

Nonsurgical Management of TMJ Disorders

- First line therapy for most TMJ conditions
- Goals of nonsurgical therapy
 - Pain reduction
 - Jaw function improvement
- Combination therapy works best
- Combination of different drug classes helps to reduce the dosage and side effects of individual medications

Treatment Options

	Comment
Diet modification	<ul style="list-style-type: none"> • Elimination of hard, chewy food • Elimination of gum chewing
Medications	<p>Five major categories</p> <ul style="list-style-type: none"> • Nonsteroidal anti-inflammatory drugs (NSAIDs): Reduces pain and inflammation; 600 mg of ibuprophen four times daily, 500 mg of naproxen twice daily • Steroids: Reduces inflammation; Medrol Dosepak (Pfizer) • Muscle relaxants: Reduces muscle spasm and tension; 5 to 10 mg cyclobenzaprine three times daily • Antidepressants: Reduces muscle tension; 10 mg amitriptyline four times daily • Anxiolytics: Reduces muscle tension; 0.25 mg alprazolam twice daily or 5 mg diazepam four times daily
Occlusal appliances	<p>Three types</p> <ul style="list-style-type: none"> • Stabilization splint: Hard acrylic splint with flat, nonguiding surface and full coverage of maxillary dentition; prevents tooth grinding but not clenching • Modified Hawley appliance: Hard acrylic maxillary appliance that allows contact of only the six anterior teeth and separates the posterior teeth; prevents both tooth grinding and clenching and is worn only at night • Repositioning splint: Hard acrylic appliance with full coverage of maxillary dentition and an inclined plane to guide the mandible to a more anterior position; used in patients when anterior mandibular positioning can recapture an anteriorly displaced disc; must be monitored carefully to prevent shifting of the dentition
Physical therapy	<ul style="list-style-type: none"> • Exercise therapy <ul style="list-style-type: none"> – Gentle range of motion exercises – Spray and stretch • Thermal therapy <ul style="list-style-type: none"> – Application of moist heat directly to the affected area for 20 to 30 minutes 4 to 6 times per day; should be combined with muscle massage • Electrogalvanic stimulation • Ultrasound
Stress-reduction techniques	<ul style="list-style-type: none"> • Relaxation techniques (ie, biofeedback, relaxation therapy audiotapes, meditation) • Stress management counseling

Arthritis of the TMJ

Noninflammatory	Inflammatory
Osteoarthritis (OA)	Rheumatoid arthritis (RA) Spondyloarthropathies Crystal-induced arthropathy Infectious arthritis

Comparison Between OA and RA

	OA	RA
Age	Later in life	Any time in life
Speed of onset	Slow, over years	Relatively rapid, weeks to months
Prevalence of TMJ involvement	8% to 16%	50%
Joint symptoms	Ache but little or no swelling	Painful, swollen, and stiff
Common joint involvement	<ul style="list-style-type: none">• Random• Single joint involvement is common	<ul style="list-style-type: none">• Symmetric joint involvement• Multiple joint involvement is common
Morning stiffness	< 1 hour	> 1 hour
Systemic effects	Not present	Present

Osteoarthritis (OA)

Demographics	<ul style="list-style-type: none"> • Most common type of arthritis involving the TMJ • Two types <ul style="list-style-type: none"> – Primary <ul style="list-style-type: none"> ◦ Idiopathic, wear and tear ◦ Older population – Secondary <ul style="list-style-type: none"> ◦ Caused by trauma, hypermobility, internal derangement ◦ Younger population (age 20 to 40)
Clinical findings	<ul style="list-style-type: none"> • Usually unilateral joint involvement • Most symptoms are localized • Primary OA: Mild discomfort • Secondary OA <ul style="list-style-type: none"> – TMJ pain increased by function – Limited mouth opening – Occasional clicking or popping sound – Crepitation may be noted in the late stages
Imaging findings	<ul style="list-style-type: none"> • CT scan is the preferred modality • Typical radiographic findings <ul style="list-style-type: none"> – Subchondral sclerosis in mandibular condyle – Condylar flattening and marginal lipping – Cortical erosion – Osteophyte formation – Narrowing of joint space
Diagnosis	Based on history, physical examination, and imaging
Treatment	<ul style="list-style-type: none"> • Conservative management <ul style="list-style-type: none"> – NSAIDs – Soft, nonchewy diet – Heat – Bite appliance, if there is parafunction • Surgical management – If conservative management for 3 to 6 months is not effective – If radiographic evidence of arthritis is present – Arthroplasty should only involve removal of any osteophytes and smoothing of any erosions; conserve as much cortical bone as possible

Rheumatoid Arthritis (RA)

Demographics	<ul style="list-style-type: none">• More than 50% of RA patients have TMJ involvement• Females > males
Clinical findings	<ul style="list-style-type: none">• Bilateral involvement• Pain, limited mandibular movement, and preauricular swelling• Pain and stiffness are usually worse in the morning and improve over course of the day• Late sequela<ul style="list-style-type: none">– Condylar resorption– Class II malocclusion– Anterior open bite– Fibrous or bony ankylosis
Imaging	<ul style="list-style-type: none">• Bilateral joint involvement in 50% to 80% of cases• Demineralization, condylar flattening, and erosion (condyle and sometimes the glenoid fossa)• Loss of ramus height• Only posterior tooth occlusion
Diagnosis	<ul style="list-style-type: none">• Based on clinical, physical, and radiographic findings• Laboratory findings<ul style="list-style-type: none">– Positive rheumatoid factor (RF), antinuclear antibodies (ANA), human leukocyte antigens (HLA) Dw5 and DRw– Elevated erythrocyte sedimentation rate– Decrease in serum albumin– Synovial aspirate: Cloudy, reduced viscosity, white cell blood count > 20,000
Treatment	<ul style="list-style-type: none">• Disease management: NSAIDs, short-term steroids, soft diet, physical therapy, immunosuppressive drugs (rheumatology referral)• Structural deformity: TMJ and orthognathic surgery

Juvenile Rheumatoid Arthritis (JRA)

Demographic	<ul style="list-style-type: none"> • Age of onset: 1 to 12 years • Females > males • 41% TMJ involvement
Clinical findings	<ul style="list-style-type: none"> • Bilateral TMJ involvement • Pain, limited mandibular movement, and preauricular swelling • Progressive Class II malocclusion and apertognathia due to loss of ramal height secondary to condylar destruction (“bird face deformity”), ankylosis in late stages
Imaging	<ul style="list-style-type: none"> • Condylar cortical bone erosion, disc thinning, loss of ramus height • Increased antegonial notching in late stages
Diagnosis	<ul style="list-style-type: none"> • Based on clinical, physical, and radiographic findings • Laboratory findings: Positive RF in 20% of patients, ANA in 60% to 80% of patients, elevated erythrocyte sedimentation rate
Treatment	<ul style="list-style-type: none"> • Disease management: NSAIDs, soft diet, physical therapy, disease-modifying antirheumatoid drugs (rheumatology referral) • Structural deformity: TMJ and orthognathic surgery when disease controlled

Spondyloarthropathies

- A disease of the vertebral column
- Associated with HLA-B27 antigens
- Seronegative for RF and ANAs
- Back pain is the most common clinical presentation of the disease
- TMJ involvement varies

Psoriatic arthritis	<ul style="list-style-type: none"> • Occurs in a third of patients with cutaneous psoriasis • Negative RF • Clinical triad <ul style="list-style-type: none"> – Psoriasis – Radiographic evidence of erosive arthritis – Negative serologic test for RF • Treatment: NSAIDs, steroids, disease-modifying drugs
Ankylosing spondylitis (Marie-Strumpell disease)	<ul style="list-style-type: none"> • A third of patients have TMJ involvement • Males > females • TMJ onset can occur several years after disease onset • Negative RF, HLA-B27 antigen positive • Treatment usually medical: NSAIDs, physical therapy, steroids, disease-modifying drugs

(Spondyloarthropathies cont)

Reactive arthritis (Reiter syndrome)	<ul style="list-style-type: none">• Clinical triad<ul style="list-style-type: none">– Arthritis (cannot climb)– Uveitis (cannot see)– Urethritis (cannot pee)• Triggered by<ul style="list-style-type: none">– Intestinal infection (<i>Salmonella</i>, <i>Shigella</i>)– Sexually transmitted disease (<i>Chlamydia</i>, gonorrhea)• Treatment: NSAIDs, steroids, disease-modifying drugs, appropriate antibiotics if specific triggering organism is identified
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Other Arthritic Diseases

Still disease	<ul style="list-style-type: none">• Subtype of JRA• Mostly boys under 5 years of age• Seronegative for both RF and ANA• Joint involvement with high fever, rash, cardiopulmonary involvement, lymphadenopathy, hepatosplenomegaly
Infectious arthritis	<ul style="list-style-type: none">• Local etiology: Trauma, TMJ surgery, extension of adjacent infection in mandible, ear• Systemic etiology (hematogenous spread): Gonorrhea, syphilis, tuberculosis, actinomycosis• General findings<ul style="list-style-type: none">– Fever, chills, swelling, regional lymphadenopathy– Painful, tender joint– Preauricular skin is warm and erythematous– If severe, malocclusion (posterior open bite on affected side or mandibular deviation) due to increased joint fluid• Lab findings<ul style="list-style-type: none">– Leukocytosis– TMJ aspirate culture: Most common microorganism is <i>Staphylococcus aureus</i>• Treatment: Incision and drainage, intravenous antibiotics

Crystal-Induced Arthropathy

- A spectrum of inflammatory arthritides that are induced by cellular reaction to crystal deposition in and around the joint space
- More common in the extracranial joints
- Two types
 - Gout: Monosodium urate monohydrate crystals
 - Pseudogout: Calcium pyrophosphate dehydrate crystals
- **Clinical presentation**
 - Trismus
 - Mandibular deviation on mouth opening
 - Preauricular pain and swelling
 - Pain in other joints (metatarsophalangeal, wrist, knee, and elbow)

- Work-up
 - Panoramic radiograph and CT scan
 - Laboratory test: Serum uric acid level
 - Joint fluid aspiration: Polarized light microscopy evaluation for crystals
- **Treatment**
 - Prophylaxis: Low purine diet, colchicine
 - Acute attack: Steroid, colchicine, NSAIDs
 - Surgery if conservative management fails
 - Arthrocentesis, arthroscopy
 - Surgical debridement and arthroplasty

Gout versus pseudogout

	Gout	Pseudogout
Etiology	Overproduction or underexcretion of uric acid	Abnormally high concentration of inorganic pyrophosphate (PPi) in synovial fluid
Risk factors	<ul style="list-style-type: none"> • Purine-rich diet • Diuretics • Excessive alcohol use 	Systemic disorders <ul style="list-style-type: none"> • RA • Hypothyroidism • Hyperparathyroidism • Familial hypocalciuria and hypercalcemia
Sex	Males > females	Females > males
Age	40+	60+
Crystal	Monosodium urate monohydrate	Calcium pyrophosphate dehydrate (CPPD)
Frequency	More common	Less common
Imaging	Nonspecific finding of chronic destructive arthropathy and intra-articular calcification	
Lab findings	Elevated serum uric acid	N/A
Polarized light microscopy	<ul style="list-style-type: none"> • Needle-shaped crystals • Negative birefringence 	<ul style="list-style-type: none"> • Rhomboid/rod-shaped crystals • Weakly positive birefringence
Locations	Small joints of hands, feet, wrists, knees, elbows	<ul style="list-style-type: none"> • Triangular ligament of wrist • Meniscus of knee • TMJ
Prophylaxis	<ul style="list-style-type: none"> • Purine-low diet • Colchicine • Urate-lowering drugs (xanthine-oxidase inhibitors, uricosuric drugs) 	Colchicine

Internal Derangements of the TMJ

Disruption of the internal aspects of the TMJ in which an abnormal relationship exists between the disc, condyle, fossa, and articular eminence.

- Characterized by
 - Disc displacement with or without reduction to normal position on mouth opening
 - Perforation of the retrodiscal tissue or articular disc
 - Degenerative changes in the disc and the articulating surfaces
- Etiology of TMJ internal derangement
 - Trauma
 - Bruxism causing altered joint lubrication and degenerative changes in the articular surfaces (friction)
 - Joint laxity

Intra-articular Changes

- Disc displacement
 - Anterior: 45%
 - Medial rotary: 29%
 - Anterolateral: 11%
 - Other (lateral rotary, lateral, medial, and posterior): 15%
- Chondromalacia
 - Softening of articular cartilage
 - Grading of chondromalacia (see page 348)
- Synovitis: Caused by TMJ overload leading to release of prostaglandin E₂, leukotriene B₄ by synovial membrane, leading to vasodilation of superficial capillaries/hyperemia

Wilkes Classification of Internal Derangements

- Joint conditions can be staged based on the Wilkes classification
- Classification based on findings from physical examination, MRI, and possible arthroscopic finding
 - Characteristics of the pain
 - Function—amount of mouth opening
 - Disc location/condition
 - Altered joint anatomy

	Pain	Opening	Disc location	Anatomy
Stage I	Occasional painless click	No limitation	Slightly forward	Normal
Stage II	Painful click	Intermittent locking	Moderate anterior disc displacement with reduction	Disc deformity
Stage III	Pain during function	Locked and restricted motion	Complete disc displacement without reduction	<ul style="list-style-type: none"> • Disc deformity • No bony changes or early changes
Stage IV	Continuous pain	Locked and restricted motion	Complete disc displacement without reduction	Moderate degenerative bony changes
Stage V	Severe pain	Locked and severely restricted motion	Perforation of retrodiscal tissue and possible disc perforation	Severe degenerative bony changes

Treatment

Treatment objectives for TMJ internal derangement

- Pain reduction
- Mandibular function improvement
- Stable occlusion

Treatment algorithm for painful TMJ clicking

Step 1	<ul style="list-style-type: none"> • Nonsurgical treatment of soft diet, analgesics, soporifics, bite appliance for 3 to 6 months • Objective is to eliminate pain, not the clicking • Patients with painless clicking require no treatment except a bite appliance if parafunction rather than trauma is the cause
Step 2	Arthrocentesis and/or arthroscopy
Step 3	Disc repositioning (discoplasty)

Treatment algorithm for TMJ locking

Step 1	Arthrocentesis (joint movement more important than disc position)
Step 2	Arthroscopy
Step 3	Open joint surgery (arthrotomy) <ul style="list-style-type: none"> • Disc repositioning (discoplasty) if disc salvageable • If disc not salvageable <ul style="list-style-type: none"> – Discectomy without replacement – Discectomy with interpositional material (dermis, cartilage, temporalis fascia, temporary silicone rubber [Silastic, Dow Corning])

Arthrocentesis

- **Indications**
 - Acute and chronic limitation of opening due to anteriorly displaced disc without reduction on mouth opening
 - Chronic pain associated with anterior disc displacement with reduction on mouth opening
 - Degenerative joint disease
- **Contraindications**
 - Overlying skin infection
 - Advanced fibrous ankylosis
 - Bony ankylosis
- Success rate: 80% to 85%
- Technique of superior joint space lysis and lavage
 - Inflow needle insertion (10-2 point): 10 mm forward from midtragus and 2 mm inferior to canthotragal line
 - Outflow needle insertion (20-10 point): 20 mm forward from midtragus and 10 mm inferior to canthotragal line
 - Minimum of 100 mL lavage with lactated Ringer solution or normal saline
 - Manipulation of mandible to break up adhesions and increase range of motion
 - Goal of 40 to 45 mm interincisal opening
 - Intra-articular injection: Steroid, sodium hyaluronate

Arthroscopy

- Can be both diagnostic and therapeutic
- **Indications**
 - Same as arthrocentesis
 - Biopsy of joint lesions
- **Contraindications:** Same as arthrocentesis

Diagnostic arthroscopy

- Seven anatomical areas to be examined during the arthroscopic diagnostic sweep
 - Medial synovial drape
 - Pterygoid shadow
 - Retrodiscal synovium—three zones
 - Oblique protuberance
 - Retrodiscal tissue attached to postglenoid process
 - Lateral recess of the retrodiscal synovial tissue
 - Posterior slope of articular eminence and glenoid fossa
 - Articular disc
 - Joint dynamics and mobility
 - Disc position
 - Roofing
 - Intermediate zone
 - Anterior recess

Operative arthroscopy

- Lysis and lavage
- Arthroplasty
- Removal of adhesions
- Capsular release
- Lateral pterygoid release
- Disc repositioning

Grading of synovitis

Acute synovitis index

- Type 1: Minimal vasodilation, no hyperemia
- Type 2: Moderate vasodilation, early hyperemia
- Type 3: Considerable vasodilation, moderate hyperemia
- Type 4: Total hyperemia, completely obliterates vascular patterns

Chronic synovitis

- Synovial hyperplasia and formation of tissue folds, particularly in the retrodiscal region

Grading of chondromalacia

- Grade I: Softening of the cartilage
- Grade II: Furrowing
- Grade III: Fibrillation and ulceration
- Grade IV: Crater formation and subchondral bone exposure

Abnormal TMJ Growth Disorders

Unilateral Condylar Hyperplasia

Excessive condylar growth that leads to facial asymmetry; there are two types

- Type I: Hemimandibular hyperplasia (HH)
- Type II: Hemimandibular elongation (HE)

	HH	HE
Growth area	Increase in total mandibular mass, bowing of inferior border	Increase only in mandibular length
Crossbite	None or slight	Yes
Open bite	Ipsilateral posterior open bite	Not present
Chin deviation	Moderate	Prominent

Diagnosis

Technetium-99m bone scan or serial cephalometric tracings are used to determine status of condylar growth.

Treatment

- Still growing
 - High condylectomy (5 to 6 mm superior condylar surface)
 - Orthognathic surgery when joint is no longer active
- No longer growing: Orthognathic surgery
- Consider simultaneous inferior border recontouring and genioplasty

Condylar Aplasia or Hypoplasia

Primary causes: Congenital

- Treacher-Collins syndrome (mandibulofacial dysostosis): Heterozygous for mutations in the *TCOF1* gene
- Hemifacial micosomia: Part of OMENS spectrum (categorization of phenotypic variations in orbit, mandible, ear, nerve [VII], and soft tissue from grade 0 [none] to grade 3 [severe or absence])
- Goldenhar syndrome (oculoauriculovertebral syndrome): Single mutant gene with defect in elastin and abnormal glycoprotein metabolism
- Hurler syndrome: Defects in lysosomal enzymes that degrade mucopolysaccharides
- Hallermann-Streiff syndrome (oculomandibulodyscephaly): Defect in elastin and abnormal glycoprotein metabolism

Secondary causes: Acquired

- Local factors: Trauma, infection from the mandibular bone or middle ear, irradiation
- Systemic factors: Toxic agents, JRA

Treatment

Reconstruction of maxillofacial and TMJ deformities

- Orthognathic surgery
- Distraction osteogenesis of mandibular ramus–condyle unit
- TMJ reconstruction with autologous graft (if skeletally immature) or alloplastic prosthesis (if skeletally mature)

Idiopathic Condylar Resorption

Progressive, bilateral, and symmetric condylar resorption followed by stabilization without further loss of height if resorbed down to sigmoid notch; no consistent or inciting event or etiology.

Demographics

- Females, age 15 to 35 years
- Most often teenage girl during pubertal growth spurts

Physical findings

- Generally good TMJ function but some TMJ discomfort and muscle hyperactivity during active phase of condylus
- Thinning and flattening of condylar heads
- Decrease in condylar height
- Loss of posterior facial height
- Mandibular retrusion
- Class II malocclusion with apertognathia

Work-up

Techetium-99m bone scan may be useful to determine if condylus is active.

Treatment

- Treatment is controversial
- Orthognathic surgery if condylus is inactive but may reactivate the resorption (bilateral sagittal split osteotomy [BSSO] may be associated with relapse)
- TMJ reconstruction

TMJ Hypomobility and Hypermobility

TMJ Ankylosis

- Characterized by a union of the condyle to the glenoid fossa
- Can be fibrous, partial bony, or complete bony ankylosis; also true (intra-articular) and false (extra-articular)
- Risk factors
 - Trauma: Condylar fracture, especially in children
 - Infection: Otitis media, suppurative TMJ arthritis
 - Inflammation: RA, ankylosing spondylitis
 - Surgery: Postoperative complication of TMJ surgery

Classifications of TMJ ankylosis

Topazian	Stage I: Ankylotic bone limited to condylar process Stage II: Ankylotic bone extending to sigmoid notch Stage III: Ankylotic bone extending to coronoid process
Sawhney	Type 1: Extensive fibrous adhesions around joint, condyle present Type 2: Bony fusion, especially at lateral articular surface, no fusion in the medial joint space Type 3: Bony bridge between ascending ramus of mandible and temporal bone/zygomatic arch Type 4: Joint is replaced by a mass of bone between ramus and skull base
He et al	A1: Fibrous ankylosis without a bony component A2: Bony ankylosis in lateral joint, residual condylar fragment is larger than 50% of contralateral normal condyle A3: Similar to A2 but residual condylar fragment is smaller than 50% of contralateral normal condyle A4: Complete bony ankylosis

Treatment objectives

- Restore function (mouth opening)
- Restore posterior ramal height and facial symmetry
- Prevent recurrence

Treatment options for adult ankylosis

- Gap arthroplasty
- Interpositional arthroplasty: Temporalis muscle, fascia, fat, auricular cartilage, dermis
- Reconstruction with costochondral graft, chondro-osseous iliac bone graft, alloplastic total joint prosthesis
- Aggressive postoperative physical therapy

Causes of failure

- Incomplete separation of the ankylosis
- Heterotopic bone formation

Treatment of pediatric ankylosis (Kaban protocol)

- Aggressive excision of fibrous and/or bony mass via gap arthroplasty
- Ipsilateral coronoidectomy
- Contralateral coronoidectomy if maximum intercuspal opening < 35 mm or no contralateral TMJ translation
- Lining of joint with temporalis fascia or native disc, if salvageable
- Reconstruction of ramus-condyle unit with either distraction osteogenesis (DO) or costochondral graft (CCG) and rigid fixation
- If DO, early mobilization of mandible starting day of surgery
- If CCG, early mobilization with minimal maxillomandibular fixation (not > 10 days)
- Aggressive postoperative physical therapy

Other Causes of Hypomobility

Trismus	<ul style="list-style-type: none">• Odontogenic infection• Myofascial pain• Neurologic origin: Tetanus• Hysterical trismus
Pseudo-ankylosis	<ul style="list-style-type: none">• Depressed zygomatic arch fracture• Dislocated zygomatic complex fracture• Coronoid hyperplasia (Jacob disease)• Myositis ossificans• Temporalis muscle scarring

TMJ Hypermobility

Subluxation

- A self-reducing derangement between the articulating components of the joint in which the condylar position is anterior to the articular eminence on wide mouth opening
- Cause: Looseness of the joint capsule and ligaments—overextension injury, extrinsic trauma (intubation, endoscopy), connective tissue disorder (Ehlers-Danlos syndrome, Marfan syndrome)
- **Treatment:** No specific treatment is generally indicated; instruct patient to avoid wide mouth opening; in extreme cases can be treated by capsulorrhaphy

Dislocation

- A non-self-reducing derangement between the articulating components of the joint in which the condylar process is held anterior to the articular eminence
- Causes: Posttraumatic capsular looseness, prolonged wide mouth opening, chronic subluxation, seizure disorders, Parkinsonism, drug-induced tardive dyskinesia (neuroleptics—eg, phenothiazines)
- Primary **treatment**
 - Bimanual reduction by pressing the mandibular rami downward to stretch the spastic masticatory muscles and then backward to relocate the condyles within the glenoid fossa
 - Intravenous diazepam given beforehand can ease the process
 - Limit excessive jaw movements for 1 to 2 weeks to allow the stretched tissues to heal

Nonsurgical management of recurrent dislocation

Intra-articular injections	<ul style="list-style-type: none">• Injection of (3 to 4 cc) sclerosing agent/autogenous blood into the superior joint space• Mechanism: Causes capsular fibrosis• Side effects: Postinjection swelling and pain for 2 to 3 days (relieved with NSAIDs)
Botulinum toxin injection	<ul style="list-style-type: none">• Injection of botulinum toxin (10 to 50 U) into the lateral pterygoid muscle• 1-week latency period; 2- to 3-month duration• Should not be done more often than every 12 weeks to avoid the development of antibodies

Surgical management of recurrent dislocation

Capsulorrhaphy	A portion of the lateral capsule is excised, and the capsule is then closed; a temporal fascia flap can be sutured to the lateral aspect of the capsule to further limit condylar translation
Dautrey procedure	Inferior displacement of the zygomatic arch anterior to the condyle to limit translatory movement
Eminectomy	Removal of a portion of the articular tubercle and eminence to allow the condyle to move freely
Lateral pterygoid myotomy	In patients with a persistent etiology such as a seizure disorder or Parkinsonism, this operation can be effective by preventing mandibular translation

Management of chronic persistent dislocation

- A mandibular dislocation that is not treated early can lead to a situation in which simple manual reduction is no longer possible even under general anesthesia because of the muscle contracture and severe fibrosis
- Such patients can be treated by temporal myotomy, which involves an intraoral vertical incision over the anterior border of the coronoid process and removal of the attachment of the temporalis muscle bilaterally

Neoplasms of the TMJ

- Rare condition
- Mean age: 42 years; females > males
- Classified into three groups
 - Pseudotumors: Most common (72%)
 - Benign lesions: 10%
 - Malignant lesions: 18%
- **Clinical presentation**
 - Many patients present with symptoms similar to a temporomandibular disorder (TMD)
 - Usually nonspecific symptoms
 - With large tumors, patients frequently have difficulty occluding the teeth, and the chin may deviate to the unaffected side
 - Swelling and altered occlusion—indicates neoplasm
 - Facial nerve paralysis—indicates malignancy
- **Radiographic findings**
 - Radiopacity: Pseudotumor > benign > malignancy
 - Radiolucency: Benign/malignant > pseudotumor

Pseudotumors	Benign lesions	Malignant lesions
<ul style="list-style-type: none">• Osteochondroma• Pigmented villonodular synovitis (PVS)• Synovial chondromatosis	<ul style="list-style-type: none">• Osteoma• Osteoid osteoma and/or osteoblastoma	<ul style="list-style-type: none">• Metastases• Osteosarcoma• Chondrosarcoma• Synovial chondrosarcoma• Synovial sarcoma

Pseudotumors

Osteochondroma	<ul style="list-style-type: none">• Pathophysiology: Metaplasia of condylar periosteum• Physical findings<ul style="list-style-type: none">– Joint pain and hypomobility– Lobulated bony outgrowth continuous with cortex of the condyle– Prognathic appearance with chin deviated to contralateral side– Ipsilateral posterior open bite• Treatment: Resection, with possible simultaneous orthognathic
PVS	<ul style="list-style-type: none">• Uncommon benign proliferative disorder of the synovium• Can affect the entire synovial membrane• Demographics<ul style="list-style-type: none">– Males = females– 3rd to 4th decade of life• Physical findings<ul style="list-style-type: none">– Often characterized by a sudden onset of joint pain and swelling; other times may develop slowly and be relatively asymptomatic– Immovable, nontender/mildly tender preauricular mass/swelling– Aggressive infiltrative behavior: Bony destruction seen on radiograph• Histology<ul style="list-style-type: none">– Hypertrophic pigmented synovial lining cells due to hemosiderin deposition– Plump spindle cells and multinucleated giant cells– Foamy macrophages due to high lipid content• Radiographic findings<ul style="list-style-type: none">– MRI<ul style="list-style-type: none">◦ Most sensitive and specific◦ Low signal intensity on T1 and T2 due to hemosiderin deposition◦ “Blooming” artifact due to hemosiderin in gradient echo sequence• Treatment: Complete excision with synovectomy +/- capsulectomy• Prognosis: High recurrence rate due to difficulty in removing the entire synovium; radiation therapy in recurrent cases may be helpful
Synovial chondromatosis	<ul style="list-style-type: none">• Proliferation of abnormal synovium with subsequent cartilaginous metaplasia• Abnormal synovium will eventually detach from membrane and form “joint mice”• Demographics<ul style="list-style-type: none">– Females > males– Mean age 46 years• Physical findings<ul style="list-style-type: none">– Restricted joint range of motion– Pain– Swelling• Radiographic findings<ul style="list-style-type: none">– Cartilaginous nodules can be both radiopaque or radiolucent depending on degree of calcification; MRI may be better than a CT scan for visualization• Treatment: Synovectomy and removal of joint mice• Prognosis: Recurrence is uncommon

Benign TMJ Tumors

Osteoma	<ul style="list-style-type: none">• Pathophysiology: Continuous formation of cortical and cancellous bone• Physical findings<ul style="list-style-type: none">– Two types<ul style="list-style-type: none">◦ Replacement of condyle by osteoma◦ Pedunculated osseous mass attached to condyle– Malocclusion and facial asymmetry similar to osteochondroma• Treatment: Similar to osteochondroma																
Osteoid osteoma	<ul style="list-style-type: none">• Pathophysiology: Proliferation of osteoblasts forming bony trabeculae set in a vascularized fibrous connective tissue stroma• Physical findings<ul style="list-style-type: none">– Localized preauricular pain and swelling– Radiograph: Mixed radiolucent/radiopaque areas with a sclerotic bony margin– Histology: Nidus of osteoid tissue surrounded by densely reactive bone• Differential diagnosis<ul style="list-style-type: none">– Fibro-osseous lesion– Low-grade osteosarcoma• Treatment: Excision by curettage <table><tr><th colspan="4">Osteoid osteoma (OO) versus osteoblastoma (OB)</th></tr><tr><th></th><th>Size</th><th>Symptoms</th><th>Histology</th></tr><tr><td>OO</td><td>< 2 cm</td><td><ul style="list-style-type: none">• Nocturnal pain• Relieved with NSAIDs</td><td><ul style="list-style-type: none">• Lack of giant cells• Less vascularized</td></tr><tr><td>OB</td><td>> 2 cm</td><td><ul style="list-style-type: none">• Pain not time related• Not relieved with NSAIDs</td><td><ul style="list-style-type: none">• Giant cells• Highly vascularized</td></tr></table>	Osteoid osteoma (OO) versus osteoblastoma (OB)					Size	Symptoms	Histology	OO	< 2 cm	<ul style="list-style-type: none">• Nocturnal pain• Relieved with NSAIDs	<ul style="list-style-type: none">• Lack of giant cells• Less vascularized	OB	> 2 cm	<ul style="list-style-type: none">• Pain not time related• Not relieved with NSAIDs	<ul style="list-style-type: none">• Giant cells• Highly vascularized
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OB	> 2 cm	<ul style="list-style-type: none">• Pain not time related• Not relieved with NSAIDs	<ul style="list-style-type: none">• Giant cells• Highly vascularized														

Malignant Neoplasms of the TMJ

- Most common malignant neoplasm of the TMJ is a metastatic lesion
 - Breast, kidney, lung, colon, prostate
- Sarcoma
 - Second most common malignancy in TMJ
 - Osteosarcoma is the most common type

Osteosarcoma

- Originates from the osteogenic mesenchymal matrix whose cells can form osteoid or osseous, cartilaginous, or fibrous tissue
- **Histology:** Cellular pleomorphism, mitotic activity, tumor giant cells, tumor cartilage
- Predisposing conditions
 - Osteogenesis imperfecta
 - Paget disease
 - Osteochondroma
 - Fibrous dysplasia
 - Radiotherapy
- **Physical findings:** Preauricular swelling and hypomobility of mandibular condyle
- **Radiographic findings** – Unicentric, bone-destructive lesion with indefinite margins
 - Osteolytic, osteoblastic, mixed
 - Three patterns of ossification based on CT scan
 - No ossification
 - Mottled ossification
 - Lamellar ossification
- **Treatment** – Preoperative adjuvant chemotherapy – Composite resection with 2-cm margin – Postoperative adjuvant chemotherapy (if positive surgical margin or large or high-grade osteosarcoma)
- **Prognosis**
 - Poor: 5-year survival rate ~ 50%
 - Lung metastasis is common

(Malignant Neoplasms of the TMJ cont)

Chondrosarcoma	<ul style="list-style-type: none"> • Malignant tumor characterized by the formation of cartilage rather than bone originating from the neoplastic cells • 5% to 10% of chondrosarcomas occur in head and neck sites • Synovial chondrosarcoma: Can arise from synovial chondromatosis (secondary) or de novo (primary) • Histologic findings – Proliferation of hyaline cartilage – Presence of a sarcomatous stroma that contains star-shaped, spindle-shaped, and round cells <ul style="list-style-type: none"> – Three histologic gradings according to number of mitoses, cellularity, and tumor size <ul style="list-style-type: none"> ◦ Grade I: No reported metastasis ◦ Grade II: 10% metastasis ◦ Grade III: 70% metastasis • Physical findings <ul style="list-style-type: none"> – Preauricular swelling and pain – Chronic and progressive limitation of mouth opening – Mandibular deviation on mouth opening • Radiographic findings – Irregular erosion of condyle with articular space calcification – Non-enhancing mass with flocculent calcification with or without osseous destruction • Differential diagnosis: Osteogenic sarcoma, pleomorphic adenoma, chondroma, Ewing sarcoma • Treatment: Resection with > 2- to 3-cm margin and adjuvant radiation therapy • Prognosis: 50% 5-year survival rate
Synovial sarcoma	<ul style="list-style-type: none"> • Soft tissue sarcoma arising from pluripotential mesenchymal cells, with cells resembling those in synovial membrane • Less than 10% of synovial sarcomas occur in the head and neck region • 90% have specific genetic translocation: Located between chromosome X and 18, t(x;18)(p11.2;q11.2) • Histologic findings <ul style="list-style-type: none"> – Characterized by both spindle and epithelioid cells – Three subtypes: Both cell types; single cell type; undifferentiated • Treatment: Wide surgical excision with adjuvant chemotherapy or radiation therapy • Prognosis <ul style="list-style-type: none"> – Poor with high metastasis rate – 5-year survival rate: 36% to 50%

Alloplastic TMJ Reconstruction

Indications	<ul style="list-style-type: none">• Severe arthritic conditions (OA, traumatic arthritis, RA)• Ankylosis• Revision procedures where other treatments have failed (eg, alloplastic reconstruction, autogenous grafts)• Avascular necrosis• Multiply operated joints• Large benign neoplasms• Malignancy (eg, post-tumor excision)• Severe condylar resorption
Contraindications	<ul style="list-style-type: none">• Presence of active or chronic infection• Inadequate bone to support implant• Partial TMJ reconstruction is not recommended<ul style="list-style-type: none">– Natural condyle can resorb when functioning against a metal fossa– A metal condyle will erode through the natural fossa• Known allergic reaction to any materials used in the components<ul style="list-style-type: none">– Patients with known or suspected nickel sensitivity should not have cobalt-chromium-molybdenum (Co-Cr-Mo) devices implanted because this material contains nickel• Skeletally immature patients

TMJ Prostheses

- All prostheses consist of two components
 - Mandibular condyle
 - Fossa component
- A vertical dimension of 20 mm is needed for both the condyle and fossa component
- Two major types
 - Stock prosthesis: Allows one-stage procedure—resection and reconstruction
 - Custom prosthesis: Requires two-stage procedure—resection, CT scan for custom joint fabrication, reconstruction
- Causes of failure
 - Biologic: Infection or immune response
 - Design: Prosthesis does not fit and/or is loose
 - Material: Breakage
 - Patient: Requests removal without biologic indication
 - History of multiple joint surgeries

	TMJ concepts	TMJ medical	Biomet
Type	Custom only	Stock and custom	Stock only
Fossa material	<ul style="list-style-type: none">Fossa: Pure titaniumArticular surface: Ultra high-molecular weight polyethylene	Chromium-cobalt alloy	Ultra high-molecular weight polyethylene
Condyle material	Titanium alloy	Chromium-cobalt alloy	Chromium-cobalt alloy
Comment	Most long-term clinical data	Metal-to-metal interface; risk of metalosis	Allows pseudotranslational movement

Warning

Avoid the use of a hemiprosthesis; a natural condyle functioning against a metal fossa can undergo resorption. A metal condyle functioning against a natural fossa can cause erosion of the glenoid fossa and possible penetration into the middle cranial fossa.

Complications in TMJ Surgery

Vascular injury	<ul style="list-style-type: none">8.6% in arthroscopic surgeryMost bleeding is not severe enough to alter surgical planSuperficial temporal artery<ul style="list-style-type: none">Within 1 mm of the arthroscopic puncture siteInjury is uncommon due to “push-away” effect during instrumentationInternal maxillary artery<ul style="list-style-type: none">Crosses medial to the condylar neck and sigmoid notch20 mm below the condylar headTreated by embolization with interventional radiologyPterygoid plexus of veinsMiddle meningeal artery
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(Complications in TMJ Surgery cont)

Nerve injury	<ul style="list-style-type: none"> • Cranial nerve (CN) V – Uncommon injury by direct traumatization – Most likely due to <ul style="list-style-type: none"> ◦ Swelling from extravasation of irrigation fluid ◦ Skin flap elevation in open surgery – Nerves prone to injury <ul style="list-style-type: none"> ◦ Auriculotemporal ◦ Inferior alveolar • CN VII – Anatomy <ul style="list-style-type: none"> ◦ Crosses zygomatic arch 0.8 to 3.5 cm (average 2.0 cm) from anterior concavity of external auditory meatus ◦ Deepest location: Preauricular region ◦ Most superficial location: 5 cm from parotid border – Incidence rate: 1% to 25%—most are transient (3 to 6 months) – From open surgery: Excessive retraction – From arthroscopic procedure: Rotational instrumentation technique rather than straight puncture motion helps to decrease nerve injury – Temporal > zygomatic branch
Infection	<ul style="list-style-type: none"> • Relatively uncommon in non-implant-related TMJ surgery • Implant-related surgery (1.34%) <ul style="list-style-type: none"> – Early: Surgical contamination (uncommon) – Late: Biofilm formation—causative species <ul style="list-style-type: none"> ◦ <i>Staphylococcus epidermis</i> ◦ <i>Staphylococcus aureus</i> ◦ <i>Pseudomonas aeruginosa</i> ◦ <i>Enterococcus</i> ◦ <i>Candida</i> – Mercuri management protocol <ul style="list-style-type: none"> ◦ Removal of device ◦ Application of antibiotic bone cement spacer
Otologic complications	<ul style="list-style-type: none"> • 0.3% to 8% of arthroscopic procedures • Types of complications (from most common to least common) <ul style="list-style-type: none"> – Blood clots in external auditory canal (EAC); prevent by packing the EAC during arthroscopic procedures – Laceration of EAC – Ear fullness sensation – Vertigo – Middle ear perforation and hearing loss

(Complications in TMJ Surgery cont)

Intracranial injury	<ul style="list-style-type: none"> • Rarely reported • Average thickness of glenoid fossa is 0.9 mm • More likely associated with postoperative prosthetic joint function than surgery • No cranial fossa perforation was found in any large retrospective arthroscopy studies
Malocclusion	<ul style="list-style-type: none"> • Arthroscopic procedure: Temporary effect due to intracapsular swelling • Open joint surgery – Due to loss of ramus height – Clinical findings <ul style="list-style-type: none"> ◦ Premature posterior contact (ipsilateral) ◦ Bilateral crossbite ◦ Anterior open bite (bilateral joint surgery) – Management, based on severity, most likely involves <ul style="list-style-type: none"> ◦ Orthodontic treatment ◦ Prosthodontic management ◦ Possible orthognathic surgery
Material failure	<ul style="list-style-type: none"> • Foreign body reaction • Loosening of hardware • Component breakage • Fixation failure
Broken instruments	<ul style="list-style-type: none"> • Associated with <ul style="list-style-type: none"> – Inexperienced surgeon – Forced and repetitive instrumentation • May require open joint surgery for removal
Heterotopic bone formation	<ul style="list-style-type: none"> • Major cause of re-ankylosis • Risk factors <ul style="list-style-type: none"> – Male – High body mass index – Low preoperative mandibular range of motion – Long duration of operation – Possible genetic predisposition • Possible preventive measures <ul style="list-style-type: none"> – Minimal 2.5- to 3.5-cm gap arthroplasty – Postoperative indomethacin use – Low-dose radiation postoperatively – Fat graft placement in the operative site
Other	<ul style="list-style-type: none"> • Scuffing of condylar cartilage (care with trocar insertion) • Extravasation of irrigation fluid (excessive perforation of capsule during needle or trocar insertion)

Recommended Readings

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Craniofacial Surgery

Jennifer Woerner and Ghali E. Ghali

- ▶ Cleft Lip and Palate
- ▶ Branchial Arch Syndromes
- ▶ Craniosynostosis
- ▶ Syndromic Craniosynostosis
- ▶ Miscellaneous Disorders

Cleft Lip and Palate

Incidence	<ul style="list-style-type: none">• Most common congenital craniofacial abnormality• Occurs in 1:700 live births, worldwide• Incidence by ethnicity among US births<ul style="list-style-type: none">– Whites 1:1,000– Blacks 1:2,000– Asians 1:500• Isolated cleft palate 1:2,000 live births; not ethnically influenced												
Differences in frequency	<ul style="list-style-type: none">• Isolated cleft lip accounts for 32% of deformities• Cleft lip and palate (CLP) accounts for 68% of deformities• In cleft lip +/- cleft palate, the ratio of males to females is 2:1• In isolated cleft palate, ratio of females to males is 2:1												
Genetics	<ul style="list-style-type: none">• Most cases of unilateral cleft lip and palate are random, nonsyndromic, isolated defects<ul style="list-style-type: none">– Not a single gene abnormality but multifactorial– Increased risk of cleft lip/palate associated with various syndromes– Isolated cleft palate patient has increased likelihood of having an associated syndrome• Risk of CLP increases with the number of parents and siblings that have a cleft deformity <table><tr><th>Family makeup</th><th>Risk of cleft lip/palate</th><th>Risk of cleft palate</th></tr><tr><td>One affected sibling or parent</td><td>4%</td><td>2.5%</td></tr><tr><td>Two affect siblings</td><td>9%</td><td>7%</td></tr><tr><td>One sibling and one parent</td><td>16%</td><td>15%</td></tr></table>	Family makeup	Risk of cleft lip/palate	Risk of cleft palate	One affected sibling or parent	4%	2.5%	Two affect siblings	9%	7%	One sibling and one parent	16%	15%
Family makeup	Risk of cleft lip/palate	Risk of cleft palate											
One affected sibling or parent	4%	2.5%											
Two affect siblings	9%	7%											
One sibling and one parent	16%	15%											
Embryology	<ul style="list-style-type: none">• 6 weeks gestation: Median nasal prominence fuses with lateral nasal prominences and maxillary prominences to form base of the nose, nostrils, upper lip, and anterior maxillary alveolus<ul style="list-style-type: none">– Disruption during this time creates clefts of the lip +/- maxilla (primary palate)• 8 to 12 weeks gestation: Palatal shelves elevate and fuse with the septum to form secondary palate<ul style="list-style-type: none">– Unilateral cleft palate occurs if one palatal shelf fails to fuse– Bilateral cleft palate occurs if both shelves fail to fuse with the nasal septum												
Classification	<ul style="list-style-type: none">• Clefting can affect lip, nose, alveolus (primary palate), and/or secondary palate (Fig 11-1)• Unilateral versus bilateral<ul style="list-style-type: none">– Clefting of the lip, premaxilla, or palate can occur on one or both sides• Complete versus incomplete<ul style="list-style-type: none">– A complete cleft lip occurs when the deformity extends into the nasal cavity– A complete cleft palate occurs when the cleft extends through both the primary and secondary palate• Submucous cleft palate<ul style="list-style-type: none">– Triad of bifid uvula, absence of posterior nasal spine, and diastasis of the levator veli palatini muscle along the midline– Often diagnosed when the child develops velopharyngeal insufficiency• Bifid uvula<ul style="list-style-type: none">– Mildest form of a palatal cleft– Affects 2% of the US population												

(Cleft Lip and Palate cont)

Common issues in CLP patients	<ul style="list-style-type: none"> • Feeding <ul style="list-style-type: none"> – May require specialized bottles (eg, Cleft Lip/Palate Nurser [Mead Johnson], Special-Needs Feeder [Medela]) or nipple modifications (eg, cross cuts) to assist in feeding – Child should be held in an upright 45-degree position while feeding – Frequent burping required due to increased air swallowing • Ear infection <ul style="list-style-type: none"> – Abnormal insertion of levator veli palatini and tensor veli palatini muscles may cause eustachian tube dysfunction – Increased risk for otitis media • Growth restriction of midface <ul style="list-style-type: none"> – Attributed to multiple surgical procedures prior to completion of maxillary growth – ~25% will require orthognathic surgery • Speech: Velopharyngeal insufficiency (VPI) occurs in 20%
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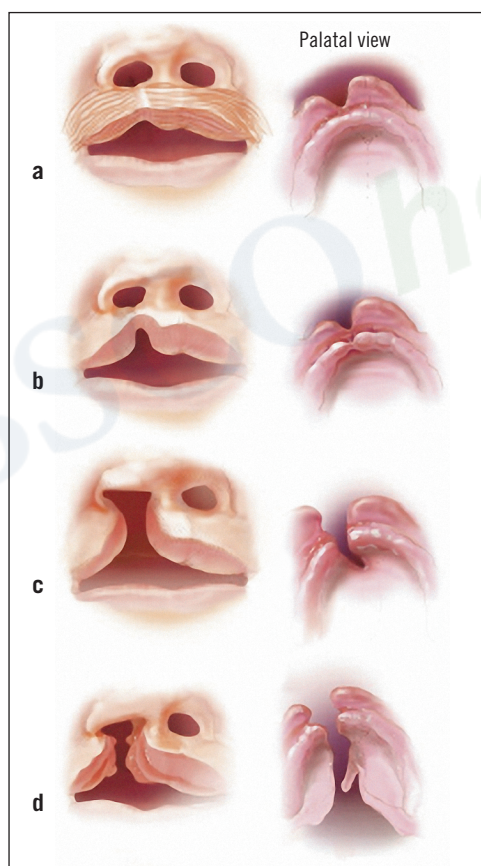


Fig 11-1 Facial and palatal views of clefting. (a) Microform cleft lip. (b) Incomplete cleft lip. (c) Unilateral complete cleft lip and alveolus. (d) Unilateral complete CLP. (Adapted with permission from Costello BJ, Ruiz RL. Unilateral cleft lip and nasal repair: The rotation-advancement flap technique. *Atlas Oral Maxillofac Surg Clin North Am* 2009;17:103–116.)

Timing of Surgical Correction

Cleft lip repair	<ul style="list-style-type: none"> • Rule of 10s <ul style="list-style-type: none"> – ≥ 10 weeks of age – ≥ 10 g/dL hemoglobin – ≥ 10 lbs
Cleft palate repair	<ul style="list-style-type: none"> • 9 to 12 months of age • 12 to 18 months of age for children with developmental delay, Pierre Robin syndrome, medical comorbidities
Pharyngeal flap (VPI management)	<ul style="list-style-type: none"> • 3 to 5 years of age but may be performed later (timing dependent on speech development)
Autogenous cancellous bone graft with BMP	<ul style="list-style-type: none"> • 4 to 6 years of age (early secondary grafting) • Performed prior to eruption of permanent incisors • Off-label use of BMP in pediatric population
Autogenous cancellous iliac crest bone graft +/- BMP	<ul style="list-style-type: none"> • Gold standard • 8 to 12 years of age (late secondary grafting) • Performed once $\frac{1}{2}$ to $\frac{2}{3}$ of the canine root is developed • Graft can be mixed with BMP prior to placement into the cleft site
Cleft orthognathic surgery	<ul style="list-style-type: none"> • Girls from 14 to 16 years of age; boys from 16 to 18 years of age • Most patients with a history of CLP have a Class III tendency, and if possible, orthognathic surgery should be delayed until skeletal maturity is reached
Cleft rhinoplasty	<ul style="list-style-type: none"> • Most commonly performed once skeletal maturity is reached • If orthognathic surgery is needed, rhinoplasty should be performed following orthognathic surgery, if possible
Lip revision	<ul style="list-style-type: none"> • Can be done at any time • If indicated in teenage patient requiring orthognathic surgery, may be beneficial to perform following orthognathic surgery
BMP, bone morphogenetic protein.	

Cleft Lip Repair

Treatment objectives	<ul style="list-style-type: none"> • Three-layered closure: Skin, orbicularis oris muscle, mucosa • Creation of a continuous sphincter with reconstruction of orbicularis oris muscle • Re-creation of normal anatomy on cleft side • Incision lines falling within natural contours of lip
Adjuvant procedures	<ul style="list-style-type: none"> • Lip adhesion <ul style="list-style-type: none"> – Performed for wide clefts or bilateral clefts with a protrusive premaxilla – Usually completed at 3 months of age; a formal cleft lip repair is completed 3 to 9 months later – May be associated with increased scarring, making it more difficult to work with the tissues at formal repair • Nasoalveolar molding devices <ul style="list-style-type: none"> – Custom made from acrylic – No research to support their use to improve esthetics of lip/palate, dental arch relationships, function, or growth – Can be bulky and difficult for infants to wear – Poor compliance
Unilateral cleft lip repair	<ul style="list-style-type: none"> • Millard advancement-rotation flap (Fig 11-2) <ul style="list-style-type: none"> – Most commonly used technique – Modified Z-plasty – Non-cleft side rotates downward to re-create philtrum – Cleft side advances the lateral lip medially to close the cleft – Pros <ul style="list-style-type: none"> ◦ Incisions concealed within natural contours of lip ◦ Can be used to close most cleft defects regardless of width ◦ Can be easily modified intraoperatively – Cons: Unmodified version may result in inadequate vertical lip length • Tennison-Randall procedure – Z-plasty technique – Pros: Advocates of procedure feel it may yield improved results in wider clefts or those requiring more vertical repositioning <ul style="list-style-type: none"> – Cons <ul style="list-style-type: none"> ◦ May not achieve adequate symmetry ◦ Incision crosses philtral column ◦ Allows little to no modification once initial

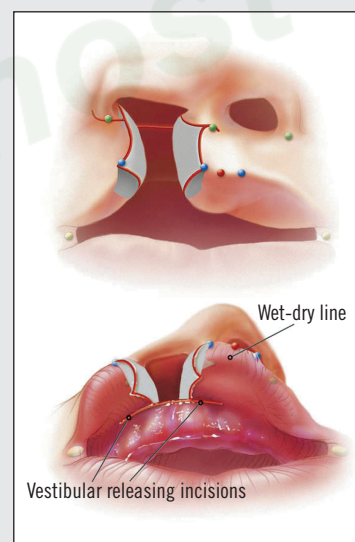
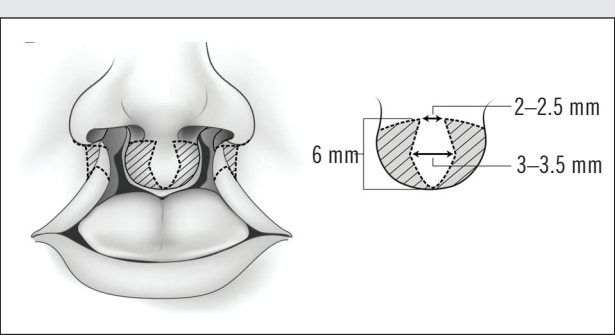


Fig 11-2 Landmarks and incision design for Millard rotation-advancement flaps for unilateral cleft lip repair. (Adapted with permission from Costello BJ, Ruiz RL. Unilateral cleft lip and nasal repair: The rotation-advancement flap technique. *Atlas Oral Maxillofac Surg Clin North Am* 2009;17:103–116.)

(Cleft Lip Repair cont)

Bilateral cleft lip repair	<ul style="list-style-type: none"> • Modified Millard rotation-advancement flap (Fig 11-3) <ul style="list-style-type: none"> – Both cleft sides repaired during procedure – Geometric design of the prolabium used to re-create the philtral column – Provides excellent symmetry of upper lip and nose – Management of premaxilla <ul style="list-style-type: none"> ◦ Presurgical taping: Gentle posterior pressure is put on the premaxilla by placing Steri-Strips (3M) across the premaxilla and taping them to the cheeks ◦ Premaxillary setback: Performed during lip repair by cutting vertically along the septum just posterior to the premaxilla and bodily moving the premaxilla posteriorly with gentle digital manipulation 	
Postoperative management	<ul style="list-style-type: none"> • Children can be fed with special nipple but may require syringe feeding during early postoperative period • Use Velcro (Velcro) arm restraints to protect repair from flailing hands/fingers • Suture line care: Cleansing with half-strength peroxide followed with bacitracin polymyx-in B ointment 	<p>Fig 11-3 Incision design for Millard rotation-advancement flaps for bilateral cleft lip repair. (Adapted with permission from Ghali GE, Ringeman JL. Primary bilateral cleft lip/nose repair using a modified Millard technique. Atlas Oral Maxillofac Surg Clin North Am 2009;17:117–124.)</p>

Cleft Palate Repair

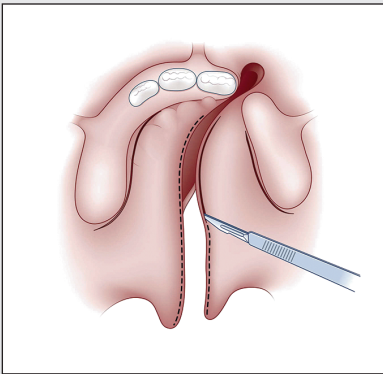
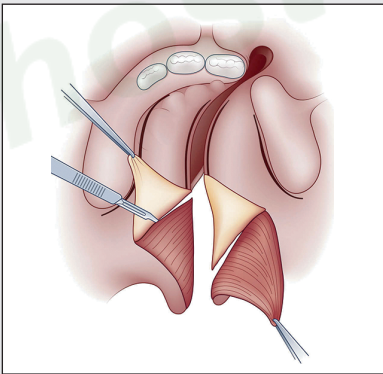
Treatment objectives	<ul style="list-style-type: none"> • Obtain three-layered closure of the soft palate and two-layered closure of the hard palate • Re-create the normal anatomy and function of the soft palate musculature, most importantly the levator veli palatini muscle • Improve velopharyngeal function 	
Cleft palate repair	<ul style="list-style-type: none"> • Von Langenbeck technique (Fig 11-4) <ul style="list-style-type: none"> – Bipedicled flaps – Levator veli palatini muscle is released from posterior hard palate and reoriented and secured horizontally within the soft palate repair – Buccal fat pad grafts can be used to fill dead space, repair perforations, or provide coverage over the denuded hard palate during the healing process – Technique can be used to close most cleft defects but does not lengthen palate • Furlow double opposing Z-plasty technique (Fig 11-5) <ul style="list-style-type: none"> – Attempts to lengthen the soft palate – May have increased fistula occurrence at the junction of the hard and soft palate • V to Y (Veau-Wardill-Kilner) flap – Variation of Von Langenbeck technique but detaches anterior pedicle <ul style="list-style-type: none"> – Attempts to lengthen palate but has never been shown to improve speech outcomes – Requires extensive dissection, which can limit midfacial growth • Bardach two-flap technique – Variation of von Langenbeck technique but detaches anterior pedicle 	
Postoperative management	<ul style="list-style-type: none"> • Arm restraints to prevent placing fingers in mouth • Patient must be on a valveless sippy cup prior to surgery • Full liquid diet for 2 weeks postoperatively, then can advance to soft, nonchewy diet • May place tongue stitch and leave for 24 hours postoperatively to prevent airway obstruction 	

Fig 11-4 Incision design for modified Von Langenbeck technique for cleft palate repair. (Adapted with permission from Smith KS, Ugalde CM. Primary palatoplasty using bipediced flaps. Atlas Oral Maxillofac Surg Clin North Am 2009;17:147–156.)

Figure 11-5 Incision design for double opposing Z-plasty for cleft palate repair. (Adapted with permission from Smith KS, Ugalde CM. Primary palatoplasty using bipediced flaps. Atlas Oral Maxillofac Surg Clin North Am 2009;17:147–156.)

Alveolar Cleft Bone Grafting

Treatment objectives	<ul style="list-style-type: none"> • Provide stability and continuity to the maxillary arch • Close oronasal fistulas • Preserve the dentition in and around the cleft site • Re-create the piriform rim
Timing	<ul style="list-style-type: none"> • Based on dental age, <i>not</i> chronologic age • Primary <ul style="list-style-type: none"> – Age 0 to 2 years – Often performed at time of primary lip repair • Early secondary – Age 2 to 5 years – Prior to eruption of incisors – Small group of surgeons advocate grafting at this time with BMP alone to avoid morbidity of second surgical site • Late secondary <ul style="list-style-type: none"> – Age 5 to 12 yrs – Performed when $\frac{1}{2}$ to $\frac{2}{3}$ of canine root has formed – Typically performed with autogenous iliac crest bone graft (AICBG) – Considered gold standard • Late <ul style="list-style-type: none"> – Age > 12 – Following eruption of canines – Typically performed with AICBG
Orthodontic considerations	<ul style="list-style-type: none"> • Grafting must be coordinated with orthodontic expansion of the maxillary arch during secondary and late grafting phases

(Alveolar Cleft Bone Grafting cont)

Grafting materials	<p>Iliac crest</p> <ul style="list-style-type: none"> • Gold standard • Pros: Provides adequate volume, proven successful, easily condensed • Cons: Second surgical site, gait disturbance, unknown effect on growth <p>Tibia</p> <ul style="list-style-type: none"> • Pros: Provides adequate volume for most clefts, bone similar quality to AICBG, ease of procedure • Cons: Second surgical site, issues with ambulation, epiphyseal injury (not recommended in growing patient) <p>Rib</p> <ul style="list-style-type: none"> • Reserved for primary repair in infants • Fallen out of favor <p>Cranial</p> <ul style="list-style-type: none"> • Pros: Less resorption, ease of procedure • Cons: Esthetic skull defect, also not commonly used <p>Allogenic</p> <ul style="list-style-type: none"> • Pros: Osteoinductive, osteoconductive, allows for eruption of teeth, no need for second surgical site • Cons: Not osteogenic <p>Alloplastic</p> <ul style="list-style-type: none"> • Pros: Osteoconductive, no need for second surgical site • Cons: Not osteogenic, teeth cannot erupt through graft <p>BMP</p> <ul style="list-style-type: none"> • Pros: No need for second surgical site, favorable soft tissue response, osteoinductive, similar bone volumes result, allows for eruption of teeth • Cons: Requires autogenous graft in older patients, moderate to severe postoperative edema associated with use, provides questionable support of ala, off-label use in pediatric patients
Procedure	<ul style="list-style-type: none"> • Buccal and palatal full-thickness mucoperiosteal sliding flaps are designed • Oronasal fistula is circumscribed, dissected, and closed, and tissue is inverted into nasal floor • Buccal and palatal flaps advanced toward alveolar cleft defect with tension relieved by circumdental sutures • Placement of graft • Closure of oral side along cleft
Postoperative management	<ul style="list-style-type: none"> • If BMP is used, it is important to remember that significant postoperative edema occurs • Sinus precautions first 3 weeks postoperatively • Liquid diet for 7 days, then may advance to soft diet • Gently brush crowns of teeth only • May use decongestant and nasal spray during postoperative period

Velopharyngeal Insufficiency Management

Diagnosis	<ul style="list-style-type: none">• Gold standard: Formal evaluation by a speech pathologist• Direct visualization using nasopharyngoscopy or videofluoroscopy may be indicated as well
Treatment objectives	<ul style="list-style-type: none">• Improved hypernasality and speech intelligibility
Surgical treatments	<ul style="list-style-type: none">• Re-repair of palate – Some advocate doing this to either lengthen the palate or confirm proper reconstruction of the levator veli palatini muscle when the primary palatoplasty was performed by another surgeon• Superiorly-based pharyngeal flap – Standard approach for surgical repair of VPI – Converts the defect of closure in the nasopharynx into two lateral ports – Advantages<ul style="list-style-type: none">◦ High success rate◦ Can easily modify surgical design in regard to width, length, and vertical position– Disadvantages<ul style="list-style-type: none">◦ Possibility of developing postoperative obstructive sleep apnea (OSA)◦ Development of hyponasality◦ Difficulty advancing maxilla during Le Fort I osteotomy• Sphincter pharyngoplasty<ul style="list-style-type: none">– Creates a single nasopharyngeal port of smaller dimension– Pro: Decreased risk of OSA– Con: Increased scarring along tonsillar pillars• Palatal augmentation – Both autogenous and alloplastic materials used in past – No longer in favor due to unpredictable outcomes, increased risk for infection, and migration of implants
Postoperative management	<ul style="list-style-type: none">• May consider keeping patient intubated postoperatively until fully awake or overnight• Tongue stitch to prevent airway obstruction• No yelling or loud vocalization for the first 2 weeks postoperatively• Liquid diet for 2 weeks postoperatively, then may advance to soft, nonchewy diet

Cleft Rhinoplasty

Characteristics	<ul style="list-style-type: none">• Medial crus of lower lateral cartilage is shorter on cleft side• Lateral crus of lower lateral cartilage is S-shaped, displaced anteriorly, and often weaker• Columella shorter on cleft side and deviated to noncleft side, with pyramid tilted toward cleft side• Turbinate hypertrophy on cleft side• Deviated caudal septum and anterior nasal spine toward noncleft side• Flattened ala• Horizontal orientation of nostril• Nasal floor is lower on cleft side or absent with bilateral clefts• Often has poor tip projection/support and bifid tip• May have nasolabial fistula
Treatment objectives	<ul style="list-style-type: none">• Correct nasal symmetry• Improve tip support and rotation• Create patent nasal airway• Improve cosmesis and facial harmony
Procedures	<ul style="list-style-type: none">• Open rhinoplasty• Commonly performed procedures<ul style="list-style-type: none">– Dorsal hump reduction– Septoplasty– Cephalic trim– Columellar strut– Intradomal sutures– Lateral osteotomies if needed to close open roof deformity– Weir excisions– Turbinectomy

Considerations in Orthognathic Surgery for the CLP patient

Intubation	<ul style="list-style-type: none">• Nasal intubation• If pharyngeal flap present, pass red rubber catheter through nose and port of pharyngeal flap first; then pass nasal RAE tube over catheter to avoid trauma to flap
Incision design	<ul style="list-style-type: none">• Unilateral CLP<ul style="list-style-type: none">– Create typical Le Fort I incision in height of vestibule– Do not extend incision posteriorly past second premolar– Use care in segmental Le Fort I not to perforate palate• Bilateral CLP – Mucosa in area of the maxillary incisors should stay intact with exception of a small stab incision to place nasal-septal osteotome<ul style="list-style-type: none">– Incisions made in height of vestibule from lateral incisors posteriorly to second premolars
Osteotomies	<ul style="list-style-type: none">• Performed like typical Le Fort I but may need to be modified to address skeletal deficiencies• Best if segmental osteotomies performed through cleft site• Must make surgical stent even for one-piece Le Fort I because maxilla may not be well consolidated and may fracture during mobilization
Amount of movement	<ul style="list-style-type: none">• Whether advancement can be achieved with Le Fort I alone or requires distraction osteogenesis is case dependent• Some surgeons advocate overcorrection of 20%• In bilateral CLP patients who have undergone multiple palatal surgeries or have significant growth restriction, may want to first consider distraction osteogenesis followed by formal orthognathic surgery
Postoperative VPI	<ul style="list-style-type: none">• Patient must be informed preoperatively that VPI is not uncommon• Should have formal speech evaluation prior to maxillary advancement• If VPI occurs, most cases will resolve on their own, but some patients will require additional speech therapy• If VPI persists may consider pharyngeal flap; surgery should be postponed for at least 6 months following maxillary advancement
RAE, right angle endotracheal.	

For a review of distraction osteogenesis, see chapter 5.

Common Secondary Cleft Lip Deformities

	Cause	Treatment
Vermilion-cutaneous mismatch	<ul style="list-style-type: none">• Improper alignment of vermillion border during primary repair• Postoperative vertical contracture of cleft side	Can be repaired with simple scar revision or Z-plasty
Whistle deformity/vertical deficiency (thin upper lip on cleft side)	<ul style="list-style-type: none">• Most common in bilateral cleft lip patients• Can be due to reduced lip volume along cleft site	<ul style="list-style-type: none">• Excision and undermining along with primary closure• Grafting with an acellular dermal matrix• V to Y advancement• Labial sulcular incision and advancement• Pedicled flaps (Abbé flap)
Lateral muscle bulging	Inadequate orbicular oris sphincter reconstruction	Take down of entire lip with re-repair and proper orientation of muscle layer

Orofacial Clefting

Tessier classification system (Fig 11-6)

- Does not explain why clefts occur in a typical pattern
- Numbered 0 to 14 and 30
- “Northbound” clefts are cephalad to palpebral fissure whereas “southbound” are caudal
- Craniofacial clefts are designated as a combination of “northbound” and “southbound” clefts

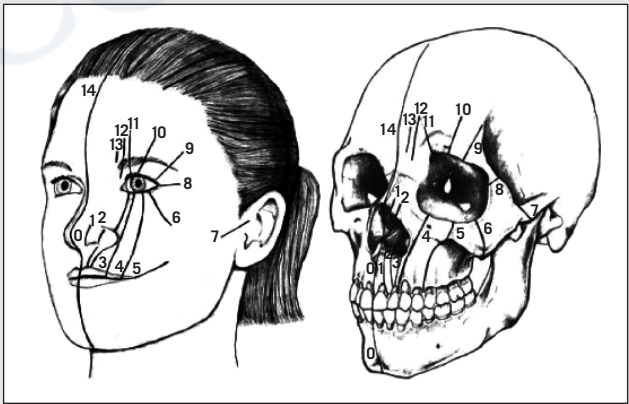


Fig 11-6 Tessier orbitocentric numbering system. (Reprinted with permission from James JN, Costello BJ, Ruiz RL. Management of cleft lip and palate and cleft orthognathic considerations. Oral Maxillofac Surg Clin North Am 2014;26:565–572.)

Branchial Arch Syndromes

Classification Systems for Syndromes Within the Oculo-Auriculo-Vertebral Spectrum

- Oculo-auriculo-vertebral (OAV) dysplasia: Ranges from hemifacial microsomia to Goldenhar syndrome
- Two major classification systems exist
 - Comprehensive system: OMENS classification
 - Mandibular only: Kaban or Pruzansky classifications

OMENS Classification

O	Orbital distortion
O ₀	Normal orbital size and position
O ₁	Abnormal size
O ₂	Abnormal position
O ₃	Abnormal size and position
M	Mandibular hypoplasia
M ₀	Normal mandible
M ₁	Small mandible and glenoid fossa with short ramus
M ₂	Short and abnormal shaped mandible
M ₃	Absence of ramus, glenoid fossa, and temporomandibular joint
E	Ear anomaly
E ₀	Normal Ear
E ₁	Mild ear hypoplasia and cupping with all structures present
E ₂	Absence of external auditory meatus with variable hypoplasia of concha
E ₃	Malpositioned lobule with absent auricle
N	Nerve involvement
N ₀	Normal facial nerve
N ₁	Upper facial nerve involvement (temporal and zygomatic branches)
N ₂	Lower facial nerve involvement (buccal, mandibular, and cervical branches)
N ₃	All branches affected
S	Soft tissue deficiency
S ₀	No apparent soft tissue and muscle deficiency
S ₁	Minimal muscle and subcutaneous deficiency
S ₂	Moderate deficiency
S ₃	Severe deficiency (muscle and subcutaneous tissues affected)

Hemifacial Microsomia

- Within the OAV syndrome spectrum
- Sporadic occurrence with variability of expression
- Due to defects in the first and second branchial arches

Common features	<ul style="list-style-type: none">• Unilateral or bilateral• Facial asymmetry due to a combination of mandibular condyle, glenoid fossa, orbit, maxilla, ear, cranial nerve, or soft tissue hypoplasia	
Classification	Kaban I	<ul style="list-style-type: none">• Mild hypoplastic state involving the muscles of mastication, the glenoid fossa, and mandibular condyle/ramus unit• Condyle has normal rotation but may have limited translation• Muscles of mastication intact• Surgical therapy not indicated
	Kaban IIA	<ul style="list-style-type: none">• Moderate hypoplasia of condyle and glenoid fossa• Medial and anterior location of the condyle in relation to the fossa• Temporomandibular joint functions well• One or more of the muscles of mastication are hypoplastic• Surgical intervention usually not indicated
	Kaban IIB	<ul style="list-style-type: none">• Moderate to severe hypoplasia of the condyle and fossa• Joint lacks articulation but still has posterior stop• No centric relation present and limited range of motion• Marked retrognathia and anterior open bite• May require reconstruction of the condyle with bone graft or mandibular osteotomies to lengthen ramus• Costochondral graft or distraction osteogenesis
	Kaban III	<ul style="list-style-type: none">• Absence of condyle/ramus• Joint lacks posterior stop• Requires reconstruction of the mandibular condyle/ramus and glenoid fossa with costochondral graft
Other considerations	<ul style="list-style-type: none">• Ear reconstruction, if needed, at age 7• Costochondral graft reconstruction of condyle between ages of 6 and 12• May require definitive orthognathic surgery on completion of facial growth• Adults may benefit from a total joint prosthesis	

Goldenhar Syndrome

Within the OAV syndrome spectrum.

Incidence	1:3,500 to 1:26,500 births
Etiology	<ul style="list-style-type: none"> • Most cases sporadic, but familial cases have been reported • Multiple chromosomal anomalies: 5q deletion, trisomy 18, 7q duplication • Some have theorized thalidomide exposure, vascular disruption of the stapelial artery, or malformation sequence with neural crest cell involvement
Common features	<ul style="list-style-type: none"> • Hemifacial microsomia • Ear malformations or microtia • Skin tags • Facial clefting • Coloboma of the eyelid • Epibulbar dermoids • Vertebral anomalies
Treatment	<ul style="list-style-type: none"> • Treat any form of facial clefting with appropriate timing • May remove skin tags and repair eyelid coloboma during infancy following ophthalmology consultation • Ear reconstruction versus temporal bone implant placement and prosthesis often considered at age 7 • Costochondral rib grafting may be required depending on severity of condyle/ramus deformity • Definitive orthognathic surgery on completion of facial growth

Treacher Collins Syndrome

Also called *mandibulofacial dysostosis*.

Incidence	1 in 25,000 to 50,000 live births
Etiology	<ul style="list-style-type: none"> • Autosomal dominant inheritance though expression is variable • Due to mutation of <i>TCOF1</i> gene

(Treacher Collins Syndrome cont)

Common features	<ul style="list-style-type: none">• Convex facial profile with prominent nasal dorsum and retrognathic mandible and chin• Antimongoloid slanting of palpebral fissures• Colobomata of lower eyelids• Hypoplasia of lower eyelids and lateral canthi• Orbital hypoplasia and/or dystopia• Absent or misshapen ears• May have hearing impairment• Hypoplasia of malar bones with clefting• Deformity of the glenoid fossa• Maxillary and mandibular hypoplasia• Hypoplasia of temporomandibular joints, muscles of mastication and facial expression• Class II malocclusion with anterior open bite and steep occlusal plane• Cleft palate and/or cleft lip may be seen• Possible choanal atresia• Various dental anomalies
Treatment	<ul style="list-style-type: none">• Ensure security of airway immediately following birth through positioning, tongue-lip adhesion, mandibular distraction, or tracheotomy• Repair of CLP or choanal atresia during infancy, if present• Orbitozygomatic reconstruction to include grafting of malar region and lateral canthopexies after age 7• Ear reconstruction with cartilage grafting or placement of temporal bone implants after age 7• Severe mandibular hypoplasia may need to be addressed during early childhood (after age 7) with either a bilateral sagittal split osteotomy or distraction osteogenesis• Kaban III condyle/glenoid fossa deformities may include glenoid reconstruction during time of malar grafting; may also require costochondral grafting• Orthognathic surgery once skeletal maturity reached• Following orthognathic surgery, may correct nasal deformity with rhinoplasty• Soft tissue and/or fat grafting may also be indicated

Nager Syndrome

Incidence	Rare, unknown incidence
Etiology	Autosomal recessive
Common features	<ul style="list-style-type: none">• Similar to Treacher Collins syndrome• Cleft palate• Hypoplasia or agenesis of the radius, thumbs, and one or more metacarpals• Lower eyelid colobomas not frequent• Short stature• Mental retardation
Treatment	Simliar to Treacher Collins syndrome

Mobius Syndrome

Incidence	Very rare, incidence unknown
Etiology	<ul style="list-style-type: none"> • Most cases sporadic but some cases familial • Mutations affecting chromosomes 1, 3, 10, and 13 have been identified
Common features	<ul style="list-style-type: none"> • Complete or partial facial diplegia: “Masklike facies” • Paralysis of other cranial nerves including III, IV, V, VI, VII, IX, X, and XII • Involvement of cerebellum, hypothalamus, or pituitary gland • Ocular deformities: Epicanthal folds, strabismus, nystagmus, ptosis • Micrognathia • Tongue abnormalities • Limb malformations • Other skeletal abnormalities • Mental retardation or autism
Treatment	No definitive treatment available, surgery symptomatic or cosmetic

Pierre Robin Sequence

Etiology	Intrauterine forces inhibit normal mandibular development during 7 to 10 weeks gestation, resulting in posterior tongue development and inability of the palatal shelves to fuse
Common features	<ul style="list-style-type: none"> • Triad of micrognathia, glossoptosis, and large U-shaped cleft palate • May present with significant airway obstruction • Can be isolated or syndrome related; Stickler syndrome (hereditary progressive arthro-ophthalmopathy) in 20% to 25% of cases
Treatment algorithm	<ol style="list-style-type: none"> 1. Observation <ul style="list-style-type: none"> – Reserved for mild disease – Mandibular growth typically improves during first year of life and catches up by age 10 2. Prone positioning 3. Douglas procedure: Lip-tongue adhesion 4. Mandibular distraction 5. Tracheotomy: Last resort; should attempt other procedures first
Other	<ul style="list-style-type: none"> • Repair of cleft palate often delayed until 12 to 18 months of age • Maximize growth and maturity of airway prior to surgery

Binder Syndrome

Etiology	<ul style="list-style-type: none">• Rhinoccephalic dysplasia• Autosomal recessive with incomplete penetrance
Common features	<ul style="list-style-type: none">• Fossa prenasalis• Hypoplasia of alar rims• Absent anterior nasal spine• Low-set and flat nasal tip• Acute nasolabial angle
Treatment	<ul style="list-style-type: none">• Bone and/or cartilage grafting of piriform rims, maxilla, or nose• Advancement of anterior nasal spine• Orthognathic surgery

Craniosynostosis

- Definition: Premature fusion of one or multiple cranial sutures resulting in characteristic abnormal head shape
- Most nonsyndromic but can be associated with various craniofacial syndromes

Incidence	Worldwide 1:1,000 live births
Etiology	<ul style="list-style-type: none">• With exception of metopic synostosis, most instances are isolated, nonsyndromic episodes that occur in utero; 5% of metopic synostoses have positive family history• Fibroblast growth factor receptor 3 gene may be involved in some cases of unilateral coronal synostosis• Sagittal synostosis more common in males; ratio of males to females is 3:1• Virchow law: Premature fusion of the suture results in arrested growth perpendicular to the suture line
Evaluation	<ul style="list-style-type: none">• Is the patient meeting developmental milestones?• Clinical examination<ul style="list-style-type: none">– Palpation of skull for movement, patent sutures, ridging, and fontanelles– Observation of head shape• Radiographic examination<ul style="list-style-type: none">– Conventional radiographs including skull films and lateral cephalograms used previously– Computed tomography of head without contrast and possible 3D reconstruction now standard of care
Indications for surgery	<ul style="list-style-type: none">• Intracranial hypertension• Neuropsychiatric disorder• Visual impairment• Psychosocial considerations

Isolated Craniosynostosis

Classification (in order of frequency)	<ul style="list-style-type: none"> • Sagittal (Fig 11-7a) <ul style="list-style-type: none"> – “Scaphocephaly” – Most common type, 1:5,000 live births – Males > females; 3:1 • Unilateral coronal (Fig 11-7b) <ul style="list-style-type: none"> – “Anterior plagiocephaly” – 1:10,000 live births – May be associated with mutation in fibroblast growth factor receptor 3 gene – Associated with defect in cranial base resulting in orbital dystopia, harlequin eye on affected side, ipsilateral midface hypoplasia, and nasal deformity • Metopic (Fig 11-7c) <ul style="list-style-type: none"> – “Trigonocephaly” – 1:15,000 live birth – More common in boys, 5% familial • Lambdoid <ul style="list-style-type: none"> – Posterior plagiocephaly – Extremely rare, 1:150,000 live births • Bilateral coronal (Fig 11-7d) <ul style="list-style-type: none"> – “Brachycephaly” – Bilateral shortening of skull base – Increased likelihood of associated syndrome if midface hypoplasia evident
Timing of reconstruction	<ul style="list-style-type: none"> • Most repairs completed prior to 1 year of age <ul style="list-style-type: none"> – Calvarium more malleable – Large bony defects able to regenerate – Continued head growth able to mold head shape • Ideal age is controversial

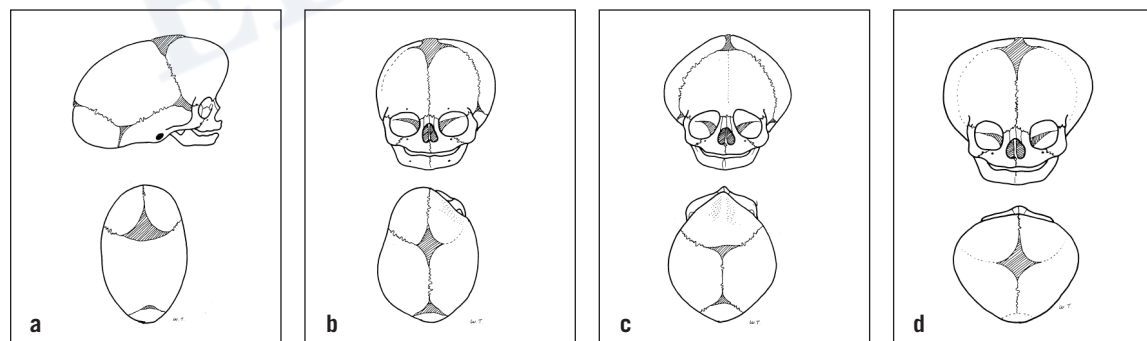


Fig 11-7 (a) Scaphocephaly. (b) Anterior plagiocephaly. (c) Trigonocephaly. (d) Brachycephaly. (Reprinted with permission from Ghali GE, Sinn DP, Tantipawasasin S. Management of nonsyndromic craniosynostosis. *Atlas Oral Maxillofac Surg Clin North Am* 2002;10:1–41.)

Surgical Correction

- Treatment objectives
 - Remove fused suture
 - Correct abnormal headshape
- Can be performed in the prone or supine position depending on deformity
- Requires coordinated effort between anesthesia, craniofacial surgery, and pediatric neurosurgery

Open approach	<ul style="list-style-type: none"> • Coronal incision • Craniotomy to remove affected suture • Depending on deformity, may require removal, reshaping, and repositioning of anterior and/or posterior vault • Orbital bandeau may be indicated in coronal or metopic craniosynostosis
Endoscopic approach	<ul style="list-style-type: none"> • Strip craniectomy performed to remove affected suture through multiple scalp incisions • Limited ability to reshape or reposition vault, therefore must be performed in the first few months of life to achieve acceptable results • Requires cranial orthotic helmet to complete reshaping

Complications if Unrepaired

Intracranial hypertension	<ul style="list-style-type: none"> • Intracranial pressure (ICP) > 15 mm Hg • 13% incidence in single suture synostosis; 42% when multiple sutures involved • Usually evident by 1 year of age • Signs and symptoms <ul style="list-style-type: none"> – Headaches – Irritability – Difficulty sleeping – Developmental delay – Large engorged scalp veins – Papilledema – Bulging fontanelle • Radiographic signs and symptoms <ul style="list-style-type: none"> – Thin cranial cortex – Hammered metal appearance of inner cortex (Lückenschädel skull)
Poor head growth	<ul style="list-style-type: none"> • Head growth and brain growth dependent on one another • 80% of head growth complete by 2.5 to 3 years of age • Sutures must be patent in order for the brain to grow • Severity of poor head and brain growth dependent on the location and number of affected sutures
Partial to complete blindness	<ul style="list-style-type: none"> • Chronic papilledema can lead to atrophy of optic nerve • If hypertelorism also present, extraocular muscles and binocular vision may be affected
Developmental delay	<ul style="list-style-type: none"> • Also related to increased ICP • May be apparent as delay in milestones, mental retardation, or behavioral issues
Psychosocial considerations	Must be discussed with parents

Positioning Posterior Plagiocephaly Versus Lambdoid Synostosis

	Positional plagiocephaly (Fig 11-8a)	Lambdoid Synostosis (Fig 11-8b)
Etiology	Infant positioning	Genetic
Onset	<ul style="list-style-type: none"> • Not present at birth • Progressive if not treated 	<ul style="list-style-type: none"> • May present at birth • Progressive if not treated
Head shape	Parallelogram	Trapezoid
Occipital shape	Flattening	Flattening
Frontal	Ipsilateral frontal bossing	Ipsilateral frontal flattening
Ear	Ipsilateral forward displacement	Ipsilateral posterior displacement
Bony ridge	Not palpable	Palpable over lambdoid suture
Torticollis	Sometimes	No
Treatment	Tummy time (placing baby on stomach while awake and supervised) +/- cranial orthotics	Total cranial vault reshaping

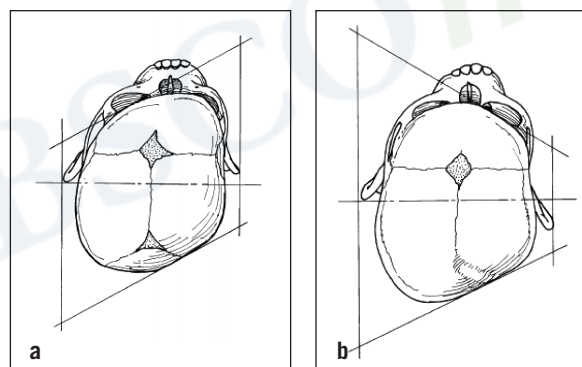


Fig 11-8 (a) Posterior plagiocephaly. (b) Lambdoid synostosis. (Reprinted with permission from Caccamese J, Costello BJ, Ruiz RL, Ritter AM. Positional plagiocephaly: Evaluation and management. *Oral Maxillofac Surg Clin North Am* 2004;16:439-446.)

Syndromic Craniosynostosis

Craniofacial dysostosis is a term used to describe syndromic forms of craniosynostosis due to involvement of the skull sutures, cranial vault, and midface.

	Affected suture	Nonfacial feature	Genetic	Mental retardation
Crouzon	<ul style="list-style-type: none">CoronalLambdoid	Rare	<ul style="list-style-type: none">Autosomal dominantSporadic	Occasional
Apert	<ul style="list-style-type: none">CoronalSquamosal	<ul style="list-style-type: none">Acne vulgarisSyndactyly	<ul style="list-style-type: none">Autosomal dominantSporadic	Common
Pfeiffer	Coronal	Syndactyly	<ul style="list-style-type: none">Autosomal dominantSporadic	Occasional
Saethre-Chotzon	Variable	<ul style="list-style-type: none">Short statureSyndactyly (soft tissue only)	<ul style="list-style-type: none">Autosomal dominantSporadic	Common
Carpenter	Variable	<ul style="list-style-type: none">ObesityHypogonadismSyndactylyVentricular septal defect/ atrial septal defect	<ul style="list-style-type: none">Autosomal recessiveSporadic	Common

Crouzon Syndrome

One of the craniofacial dysostosis syndromes with associated defect affecting cranial base.

Genetics	Autosomal dominant inheritance, multiple mutations to <i>FGFR2</i> gene
Common features	<ul style="list-style-type: none">Craniosynostosis with brachycephalic head shape, typically bilateral coronalMidface hypoplasia with Class III malocclusionProptosis due to orbital hypoplasia5% have acanthosis nigricans10% hydrocephalus

(Crouzon Syndrome cont)

Treatment	<ul style="list-style-type: none">• Primary cranio-orbital decompression<ul style="list-style-type: none">– Cranial vault reshaping to release synostotic sutures along with supraorbital bandeau expansion and advancement– Performed between 6 and 12 months of age unless signs of increased ICP– May require additional cranial vault procedures for further decompression or reshaping• Midface correction<ul style="list-style-type: none">– Performed after age 7 to allow completion of orbital growth– Midface deformities can be addressed with monobloc advancement, Le Fort III osteotomies, or facial bipartition depending on dysmorphology– Residual cranial vault deformities can also be addressed at this time• Definitive orthognathic therapy<ul style="list-style-type: none">– Often patients have Class III malocclusion and anterior open bite– Often require Le Fort I osteotomy and/or segmentalization performed upon completion of skeletal growth– Must assess patient's functional and esthetic needs and perform mandibular surgery and/or genioplasty if needed
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Apert Syndrome

A craniofacial dysostosis syndrome that, due to cartilage dysplasia at the cranial base, results in a wide, flat, and retrusive midface.

Genetics	Mutation of <i>FGFR1</i> and <i>FGFR3</i>
Common features	<ul style="list-style-type: none">• May have syndactyly of hands and feet• May have fusion of other joints including elbows and shoulders• Fusion of cervical vertebrae• 2% hydrocephalus• Can have internal organ anomalies• Downward slanting lateral canthi and S-shaped upper eyelid ptosis• Acne common• Severe mental retardation also common

(Apert Syndrome cont)

Treatment	<ul style="list-style-type: none">• Primary cranio-orbital decompression<ul style="list-style-type: none">– Cranial vault reshaping to release synostotic sutures along with supraorbital bandeau expansion and advancement– Performed between 6 and 12 months of age unless signs of increased ICP– May require additional cranial vault procedures for further decompression or reshaping• Midface correction<ul style="list-style-type: none">– Performed after age 7 to allow completion of orbital growth– Midface deformities can be addressed with a facial bipartition to address the retrusive middle third of the face– Residual cranial vault deformities can also be addressed at this time• Definitive orthognathic therapy<ul style="list-style-type: none">– Often patients have Class III malocclusion and anterior open bite– Often require Le Fort I osteotomy and/or segmentalization performed upon completion of skeletal growth– Must assess patient’s functional and esthetic needs and perform mandibular surgery and/or genioplasty if needed
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Pfeiffer Syndrome

One of the craniofacial dysostosis syndromes.

Genetics	<ul style="list-style-type: none">• Can be autosomal dominant with complete penetrance• Mutations in <i>FGFR1</i> and <i>FGFR2</i>
Common features	<ul style="list-style-type: none">• Multiple suture craniosynostoses, often bilateral coronal but can have cloverleaf deformity (Kleeblattschadel)• Broad thumbs and great toe• Occasionally partial soft tissue syndactyly of hands
Treatment	Similar to Apert syndrome

Carpenter Syndrome

One of the more rare craniofacial dystostosis syndromes.

Genetics	Autosomal recessive
Common features	<ul style="list-style-type: none">• Craniosynostosis• Preaxial polysyndactyly of the feet• Short fingers with clinodactyly• Variable soft tissue syndactyly• Other: Short stature, obesity, heart defects, variable mental retardation
Treatment	Similar to Crouzon syndrome

Saethre-Chotzen Syndrome

Genetics	Autosomal dominant
Common Features	<ul style="list-style-type: none"> • Craniosynostosis • Low frontal hairline • Upper eyelid ptosis • Brachydactyly • Cutaneous syndactyly • Facial asymmetry • Other skeletal abnormalities
Treatment	Similar to Crouzon syndrome

Miscellaneous Disorders

Holoprosencephaly

- Spectrum of disorders affecting brain and face
- Common features
 - Wide bilateral CLP or median cleft lip
 - Poorly formed prolabium
 - Clefting of nose
 - Hypotelorism
 - Must rule out encephalocele of forehead and nasal region
 - May have holospheric cerebrum
- Treatment: Similar timing as in less severe CLP patients except may have to stage lip and/or palate closure due to width of cleft and unavailability of tissue

Encephalocele

Incidence	Frontonasal in 1:35,000 live births (1:5,000 in southeast Asia)
Etiology	Neural tube defect allowing the brain and dura to escape through defect in skull
Classification	<ul style="list-style-type: none"> • Occipital: 75% • Parietal: 10% to 14% • Anterior: Further subdivided into nasofrontal, nasoethmoidal, and naso-orbital
Common features	<ul style="list-style-type: none"> • Hydrocephalus • Visual problems • Mental retardation • Growth retardation • Seizures

(Encephalocele cont)

Treatment	<ul style="list-style-type: none">• Ventriculoperitoneal shunt placement if associated with hydrocephalus• Neurosurgical plication with autogenous cranial bone grafting in infancy• Definitive reconstruction of cranial vault, orbits, and nasal bones at age 4 to 7• Some patients may need orthognathic surgery or nasal reconstruction upon completion of facial growth
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Frontonasal Dysplasia

Etiology	<ul style="list-style-type: none">• Rare, X-chromosome linked• Females > males• Mutation in <i>EFNB1</i> gene
Common Features	<ul style="list-style-type: none">• Coronal synostosis• Hypertelorbitism• Limb anomalies
Treatment	<ul style="list-style-type: none">• Anterior cranial vault reshaping, if indicated• Hypertelorism correction at ~6 years of age• Rhinoplasty when nasal growth complete; may require bone grafting of dorsum

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Cosmetic Surgery

David E. Webb and Peter D. Waite

- ▶ Facial Analysis
- ▶ Upper Third Esthetic Surgery
- ▶ Middle Third Esthetic Surgery
- ▶ Lower Third Esthetic Surgery
- ▶ Ancillary Procedures

Facial Analysis

General Considerations

- What is the patient's chief complaint? What feature or aspect stands out when first examining the face?
- Overall face shape (round, oval, triangular) and asymmetries (brow, pupil, mandible, etc)
- Evaluate facial thirds from top down; evaluate individual esthetic subunits within each third

Soft Tissue Considerations

- Skin: Texture, thickness, elasticity, degree of pigmentation and sun damage (see Fitzpatrick and Glogau tables), evaluation of scarring; assess skin in animation and repose to differentiate dynamic (mimetic) from static rhytids
- Subcutaneous adipose content
- Muscular component (hypertrophy, wasting, redundancy, dehiscence)
- Dedo profile classification (see table)
- Hair considerations: Color, density, and location of brow and scalp hair are crucial when considering forehead, brow, and face-lifting procedures

Skin Evaluation

Fitzpatrick sun-reactive skin types

	Skin color	Tanning response	
		Burns	Tans
I	White	Always	Never
II	White	Usually	Difficult
III	White	Sometimes	Sometimes
IV	Brown	Rarely	Easily
V	Dark Brown	Very Rarely	Very Easily
VI	Black	Never	Always

Glogau photoaging classification

	Age (y)	Wrinkles	Makeup use	Actinic changes	Pigment changes
I (mild)	28–35	None	Rare	None	Mild
II (moderate)	35–50	In motion	Some foundation	Palpable, not visible	Early brown spots
III (advanced)	50–65	At rest	Heavy foundation	Visible actinic keratosis	Dyschromias, telangiectasia
IV (severe)	65–75	Only wrinkles	Minimal benefit (foundation cakes and cracks)	Actinic keratosis +/- skin cancer	Yellow-gray skin

Dedo facial profile classification

	Description	Surgery indicated
I (relatively normal)	Minimal neck deformity	Submental access only
II (skin laxity)	Little cervical fat or attenuated platysma	CFR to redrape skin without submental incision or liposuction
III (fat accumulation)	Good skin and platysma tone	Submental +/- periauricular access for fat removal only
IV (platysmal banding)	Laxity of all soft tissue layers	CFR to redrape skin + submental incision + platysmal plication
V (retrognathic/microgenic)	Mandibular deficiency	Genial implant versus genioplasty versus orthognathic surgery
VI (low hyoid)	"Problem neck"	Hyoid rarely repositioned; limits esthetic outcome of CFR
CFR, cervicofacial rhytidectomy.		

Think about approach to mandible: Skin (II), then fat (III), then platysma (IV), then bone (V).

Hard Tissue Considerations

- Osseous foundation
 - Overall profile: Orthognathic, retrognathic, prognathic
 - Examine osseous contour/position individually: Frontal, nasal, zygomatic, maxillary, mandibular, and hyoid bones
- Cartilaginous framework: Ear projection, nasal esthetics
- Dental contributions: Effect on nasolabial angle, tooth to lip relation, occlusion

Functional Considerations

- Myofascial pain resulting from chronic brow elevation
- Tension-type headaches resulting in chronic brow elevation
- Visual field impairment; correctable if due to brow ptosis, upper eyelid dermatochalasis, or lower lid steatoblepharon
- Nasal function: Internal nasal valve collapse may lend itself to open rhinoplasty

Upper Third Esthetic Surgery

- Superior border: Trichion (point where hairline meets the forehead)
- Inferior border: Soft tissue glabella (most prominent point of forehead between brows)

Forehead Anatomy

	Clinical significance (Figs 12-1 and 12-2)
Ideal brow position	<ul style="list-style-type: none">• Female: Arched, 2 mm above orbital rim medially, 10 mm above orbital rim at apex (coincident with lateral limbus), tail not extending past alar-canthal line• Male: Not arched, slightly above or at rim• Brow lifting considered if brow-to-pupil distance is < 2.5 cm• Average forehead length: ~5 cm as measured from trichion to soft tissue glabella• Brow ptosis: Inferiorly displaced brow position• Lateral hooding: Obscuration of the lateral aspect of the upper lid crease secondary to lateral brow ptosis rather than redundant eyelid skin
Frontalis	Only brow elevator; controls transverse brow lines (forehead rhytids)
Procerus	Brow depressor; controls transverse glabellar lines ("bunny lines" also caused by transverse nasalis)
Corrugator supercilii	Brow depressor; controls vertical glabellar lines ("11s" or "frown lines")
Depressor supercilii	<ul style="list-style-type: none">• Medial-most fibers of orbicularis oculi that lie below medial brow• Brow depressor resection or botulinum toxin injection can lead to overelevation of the medial brow ("surprised look")
Orbicularis oculi	Brow depressor; controls lateral orbital rhytids ("crow's feet")
Sentinel vein (medial zygomaticotemporal)	<ul style="list-style-type: none">• Coincides with location of temporal branch of facial nerve• Runs perpendicular to temporalis fascia 1 cm lateral to zygomaticofrontal suture• Failure to identify this "bridging" vessel can lead to bleeding during endoscopic browlift
Supraorbital nerve	<ul style="list-style-type: none">• Exits orbital rim above medial limbus; dissection must be in subperiosteal or subcutaneous plane near orbital rim to prevent long-term hypoesthesia/anesthesia• Supplies sensation to brow
Supratrochlear nerve	Exits orbital rim 9 mm medial to supraorbital nerve; supplies sensation to brow
Temporal branch of facial nerve	<ul style="list-style-type: none">• Runs deep to temporoparietal fascia• Responsible for brow motion; injury leads to brow paresis or paralysis
Scalp	<ul style="list-style-type: none">• Layers for SCALP mnemonic: Skin, subcutaneous connective tissue, aponeurosis and muscle (galea), loose areolar tissue (subgaleal fascia), and pericranium• Mnemonic only valid in central aspect of forehead• Layers for scalp temporally (between conjoint tendon and zygomatic arch): Skin, subcutaneous connective tissue, temporoparietal fascia, loose areolar tissue (subtemporoparietal fascia), temporalis fascia, temporalis muscle, pericranium

(Forehead Anatomy cont)

Conjoint tendon/ zone of fixation	Confluence of galea and pericranium (from superior) with temporoparietal fascia, temporalis fascia, and pericranium (from below) at the superior temporal line
Orbital ligament	Most anteroinferior portion of conjoint tendon where fascia fuses with superolateral orbital rim

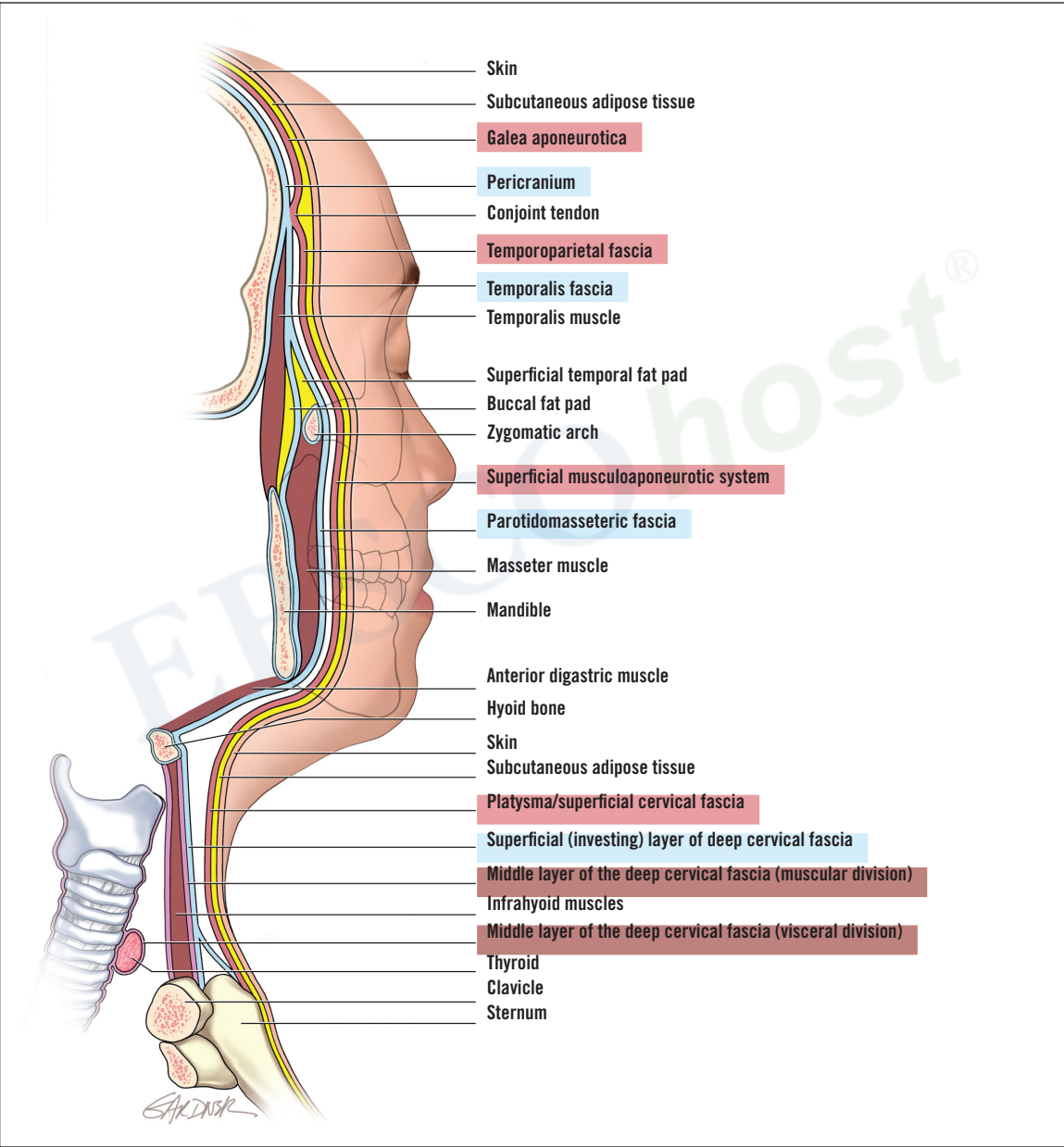


Fig 12-1 Anatomical representation of head and neck fascial planes. The superficial fascia (*maroon*) and deep fascia (superficial/investing layer, *blue*; middle layer, *purple*) of the head and neck are depicted. A surgical plane (*white layer* between the superficial and deep cervical fascia) has been added for clarity.

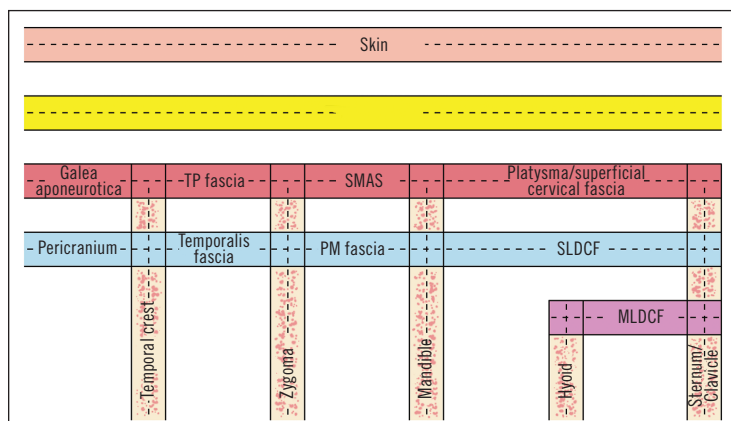


Fig 12-2 Diagrammatic “roadmap” representation of head and neck fascial planes. The superficial layer of the deep cervical fascia (SLDCF, *blue*) and the middle layer of the deep cervical fascia (MLDCF, *purple*) are continuous throughout the head and neck but change names at osseous “intersections.” Note that only the muscular division of the MLDCF is represented. TP, temporoparietal; SMAS, superficial musculoaponeurotic system; PM, parotidomasseteric.

Brow/forehead lift technique

	Incision placement	Dissection plane	Excision to lift ratio	Indications and advantages	Contraindications and disadvantages
Direct	Slightly within superior aspect of eyebrow	Subcutaneous	1:1	Thick eyebrow, direct correction of brow asymmetry	Thin, light eyebrows, bony recontouring not possible
Midforehead	Prominent transverse brow crease	Subcutaneous	1:1	Prominent transverse brow rhytids, correct asymmetry	Midforehead scar
Coronal	Well behind hairline from ear to ear	Subperiosteal versus subgaleal centrally, below temporoparietal fascia temporally	1.5–2:1	Hidden scar (if full hair), raises the hairline, bony recontouring possible	High hairline (raises hairline), long scar, scalp hypesthesia, traumatic alopecia, limited in men
Trichophytic	Slightly behind hairline centrally, arcing posterior from widow’s peak toward ear temporally	Subperiosteal versus subgaleal versus subcutaneous centrally, below temporoparietal fascia temporally	1–1.5:1	Can shorten long foreheads, can excise thinning hair-bearing areas temporally, bony recontouring possible	Potentially visible scar at the hairline
Endoscopic	4 to 5 incisions 2 cm within hairline (2 to 3 sagittal and/or parasagittal and 2 temporal)	Subperiosteal centrally, below temporoparietal fascia temporally	N/A	Hidden, short scars, minimal bony recontouring possible	High hairline, balding, excessively rounded frontal bone

Endoscopic browlift technique

1. Mark two parasagittal vertical incisions (located superior to lateral limbus for vertical vector) 2 cm behind hairline, 2 cm in length
2. Additional midline incision used if access or elevation of medial brow necessitates
3. 2-cm temporal incision placed within temporal tuft, centered on alar-canthal line (superolateral vector), marked bilaterally
4. Local anesthesia with vasoconstrictor is injected along marked incisions
5. Incise down to skull at parasagittal ports and down to temporalis fascia temporally
6. Two pockets of dissection developed: central and temporal
7. Central pocket dissected in subperiosteal plane blindly inferiorly to 2 cm above supraorbital rim, 5 to 10 cm posterior to incision, and laterally to superior temporal line
8. Temporal pockets dissected endoscopically below the temporoparietal fascia medial to superior temporal line and inferiorly until sentinel vein identified
9. Combination of direct and endoscopic access used to join lateral and central pockets (join by dissecting from lateral to medial)
10. Endoscopic dissection allows release of orbital ligament, subperiosteal elevation over orbital rims, brow periosteal scoring, and optional corrugator resection
11. Resuspend brow and secure in new position with suture (via bone tunnel or absorbable screw) or Endotine (MicroAire) fixation device (via endoscopic ports) centrally and with suture laterally
12. Close incisions with deep sutures followed by staples
13. Supportive dressing to be worn continuously for 1 to 2 weeks, then at bedtime only for 1 to 2 weeks

Middle Third Esthetic Surgery

- Superior border: Soft tissue glabella (most prominent point of forehead between brows)
- Inferior border: Subnasale (point where upper lip becomes columella)

Periorbital Anatomy (Fig 12-3)

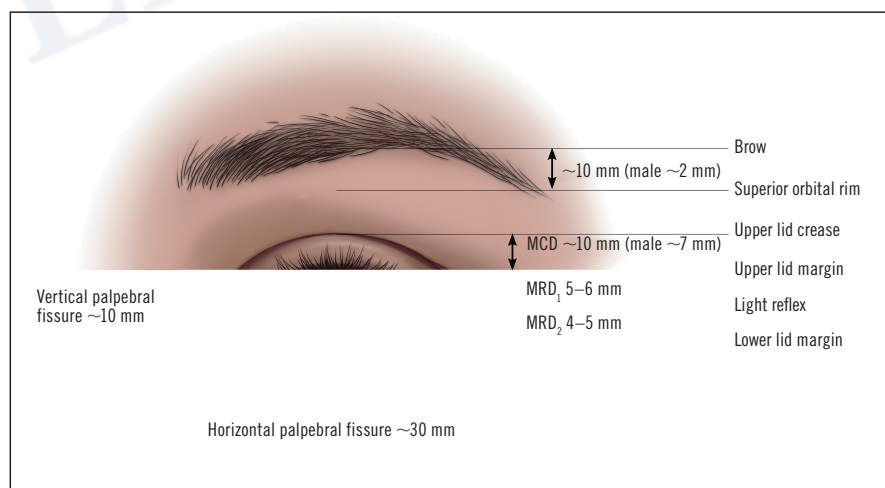


Fig 12-3 Periorbital surface anatomy. MCD, margin crease distance; MRD₁, margin reflex distance 1; MRD₂, margin reflex distance 2.

	Description	Clinical significance
Eyelid surface anatomy		
Upper eyelid crease	<ul style="list-style-type: none"> Also known as <i>supratarsal skin crease/ fold</i> and <i>superior palpebral sulcus</i> Superoinferior position is determined primarily by the insertion of the levator aponeurosis into the skin and secondarily by the location where the orbital septum merges with the levator aponeurosis 	Asian upper lids without a crease result from a lower insertion of the levator aponeurosis into the skin (just above the eyelashes) as well as a lower point of attachment of the septum to the levator aponeurosis (at the superior tarsal border versus ~10 mm above the tarsal plate in whites)
Margin crease distance	Distance from upper lid crease to lid margin during downward gaze	Normally ~10 mm in females and ~7 mm in white males
Lower eyelid crease	<ul style="list-style-type: none"> Also known as <i>infratarsal skin crease</i> and <i>inferior palpebral sulcus</i> Determined by insertion of capsulopalpebral fascia into skin 	Normally 5 mm below lid margin medially and 7 mm laterally
Margin reflex distance 1 (MRD₁)	Distance measured from the upper eyelid margin to the corneal light reflex; upper lid normally covers up to 2 mm of the upper cornea	Normally 4 to 5 mm when a light is directed at the patient's eyes (at eye level) <ul style="list-style-type: none"> If < 4 mm, lid ptosis likely exists If > 5 mm, rule out thyroid exophthalmopathy
Margin reflex distance 2 (MRD₂)	<ul style="list-style-type: none"> Distance measured from lower eyelid margin to corneal light reflex Lower lid normally lies at the corneoscleral limbus 	Normally 5 to 6 mm
Palpebral fissure: Vertical distance	From upper lid margin centrally to lower lid margin in primary gaze (normally ~10 mm)	$MRD_1 + MRD_2 = 10 \text{ mm}$ (if < 10 mm, suspect lid ptosis)
Palpebral fissure: Horizontal distance	From medial to lateral canthal angle (normally ~30 mm)	Eyelids close passively because palpebral fissure length is 3x its height (similar 3:1 dimensions used with excisional biopsy technique to achieve tension-free closure)
Lateral canthal angle	Angle formed by upper and lower eyelids (normally 30 to 40 degrees and lies 2 mm superior to medial canthal angle)	<ul style="list-style-type: none"> Antimongoloid slant: Lateral canthus lower than medial canthus (as seen in Treacher Collins syndrome) Blunted angle often represents lid laxity; warrants lid-tightening procedure, contraindication for skin only blepharoplasty as ectropion will likely result
Skin and subcutaneous tissue	Thinnest skin of the body (< 1 mm) w/ minimal subcutaneous fat; fine wrinkles readily form	<ul style="list-style-type: none"> Dermatochalasis: Excess eyelid skin Cicatricial ectropion: Postoperative ectropion after over-resection of skin Blepharochalasis: Rare, recurrent, episodic idiopathic eyelid edema, typically found in younger women <ul style="list-style-type: none"> An indication for blepharoplasty (after condition has run its course)

(Periorbital Anatomy cont)

	Description	Clinical significance
Orbicularis oculi muscle	<ul style="list-style-type: none"> Innervated by zygomatic (and temporal) branches of facial nerve Arbitrary division into orbital and palpebral parts (which are further divided into preseptal and pretarsal portions) Can become hypertrophic or redundant 	Indication for muscle removal as well as skin during blepharoplasty
Tarsal plates	<ul style="list-style-type: none"> Dense fibrous connective tissue Upper and lower tarsal plates are, respectively, 10 mm and 5 mm wide centrally 25 sebaceous meibomian glands, whose exocrine ducts enter just posterior to grey line, span the plates 	Grey line: Gap formed between pretarsal tissue and tarsal plate at lid margin that serves as the junction of skin and conjunctiva
Orbital septum	Connective tissue that arises from periosteum of bony orbit (arcus marginalis) and separates the orbit from the eyelids	Attenuated orbital septum can allow orbital fat herniation (steatoblepharon)
Upper eyelid retractors		
Levator palpebrae superioris muscle	Upper eyelid retractor that arises from lesser wing of sphenoid, runs above superior rectus muscle within orbit, passes through orbicularis oculi and inserts into preseptal skin	Primary determinant of supratarsal skin crease position; disinsertion can result in blepharoptosis
Mueller muscle	Upper lid retractor that arises from under surface of levator palpebrae superioris muscle posteriorly, firmly attaches to palpebral conjunctiva anteriorly prior to inserting into superior margin of tarsus; widens palpebral fissure ~2 mm	Sympathetically innervated smooth muscle allows pharmacologic correction (apraclonidine) of lid ptosis resulting from inappropriate botox injections
Fat pads	Upper lid has 2 fat pads (medial [nasal] and central); lower lid has 3 fat pads (medial [nasal], central, and lateral)	<ul style="list-style-type: none"> Medial and central lower fat pads separated by inferior oblique muscle; central and lateral pads separated by arcuate expansion of capsulopalpebral fascia Retroseptal hematoma can result from inadequate hemostasis after fat removal Upper eyelid compartments are pink (lateral lacrimal gland), yellow (central fat pad), and pale yellow/white (medial fat pad)
Conjunctiva	<ul style="list-style-type: none"> Palpebral conjunctiva: Lines inner surface of eyelids until the depth of the fornix Bulbar conjunctiva: Conjunctiva covering the globe 	Palpebral conjunctival cicatrix can lead to post-operative entropion

Blepharoplasty Techniques

Upper eyelid	<ul style="list-style-type: none">• Skin excision only• Skin and muscle excision• Skin, muscle and fat excision• Browpexy via upper blepharoplasty incision
Lower eyelid	<ul style="list-style-type: none">• Skin pinch (skin only); elevate excess skin by crimping, then excise• Transconjunctival (fat only), often in combination with laser resurfacing; incision 4 to 5 mm inferior to inferior tarsal margin (retroseptal) from punctum to lateral canthus• Subciliary: ~1.5 mm below the lower lashes

Upper blepharoplasty technique

1. Correct brow ptosis, if present
2. Mark upper lid crease, if present (if not apparent, place mark above the upper lid margin 10 to 12 mm in females and by 7 to 8 mm in males)
3. Determine degree of dermatochalasis by pinching the redundant skin until slight eyelid eversion
4. Mark upper border of redundant skin (this is the upper blepharoplasty incision)
5. Generally maintain 10 mm of eyelid skin below lower incision and 10 mm of brow skin above upper incision
6. If desired, place corneal protector (use topical anesthesia if surgery performed under sedation)
7. Infiltrate subcutaneously with a local anesthetic containing a vasoconstrictor
8. Incise through skin
9. Elevate skin flap from underlying orbicularis oculi muscle (alternatively, a skin-muscle flap may be elevated from the underlying orbital septum)
10. If indicated, isolated nicks in the orbital septum provide access for partial removal of fat pads
11. Gentle globe retropulsion results in fat herniation
12. Conservatively excise excess fat with electrocautery
13. Ensure hemostasis with bipolar electrocautery
14. A single resorbable suture may be placed if the septum was widely violated; otherwise skin margins are approximated using subcuticular or interrupted sutures
15. Immediate postoperative cold compresses are recommended
16. Acceptable postoperative instructions include maintaining the head above the heart at all times and no lifting over 10 pounds for 10 days

Transconjunctival lower blepharoplasty technique

1. Place corneal protector (use topical anesthesia if necessary)
2. Lower eyelid retraction requires either a DesMarres eyelid retractor or a silk retraction suture
3. Infiltrate the infratarsal conjunctiva with a local anesthetic containing a vasoconstrictor
4. Incise the palpebral conjunctiva overlying the fat pads with electrocautery (usually 4 to 5 mm below the inferior tarsal margin)
5. Retroseptal transconjunctival approach provides direct access to the fat pads without postoperative scarring or weakening the orbital septum
6. Gentle globe retropulsion results in fat herniation
7. Conservative fat excision may be performed with electrocautery
8. Ensure hemostasis with bipolar electrocautery
9. No conjunctival closure needed
10. Postoperative instructions are similar to those for upper blepharoplasty
11. Note: transconjunctival fat excision/recontouring is typically performed in conjunction with a skin resurfacing procedure (ie, laser resurfacing)

Blepharoplasty Complications

	Description	Treatment
Hematoma	<ul style="list-style-type: none">• If orbital septum breached, bleeding can lead to retroseptal hematoma (0.04% incidence)• Can cause blindness	Initially release blepharoplasty sutures in effort to drain; perform lateral canthotomy and cantholysis as needed
Lower lid retraction	Results from excessive removal of anterior lamella (skin +/- muscle) or scarring of middle lamella (septum)	<ul style="list-style-type: none">• Conservative correction: Massage, stretch• Surgical correction: Tarsal strip, lengthen posterior and/or anterior lamella with mucosal and full-thickness skin grafts, respectively
Lagophthalmos	Inability to completely close the eyelids (~2 to 3 mm); not concerning immediately postoperatively	<ul style="list-style-type: none">• Nonsurgical: Artificial tears, lubricating ointment• Surgical: Correct underlying deficiency

Rhinoplasty Anatomy

- External nasal examination: Divide nose into vertical thirds and evaluate from frontal, profile, and basal views – Frontal
 - Upper third: Brow-tip esthetic line; evaluation continues as the line transitions through middle and lower thirds
 - Middle third: Straightness of dorsum; if crooked, is it deviated or twisted? is the defect in cartilage or bone?
 - Lower third: Tip definition and bulbousness, alar-columellar relationship– Profile
 - Upper third: Radix position (projection and height)
 - Middle third: Dorsal height
 - Lower third: Tip projection and rotation, alar-columellar relationship, nasolabial angle, and chin projection– Basal
 - Straightness of dorsum, tip definition, columellar-lobule relationship, nostril symmetry, and alar base width
- Internal nasal examination: Evaluate for turbinate hypertrophy, septal deviation, and nasal valve function
 - Cottle maneuver (lateral distraction of the cheek) evaluates internal nasal valve function
 - Modified Cottle maneuver (thin instrument or cotton swab is used to independently elevate the ipsilateral upper lateral cartilage or lower lateral cartilage) differentiates external and internal valve collapse
 - Tests are positive if nasal obstruction relieved

Nasal structures

	Description	Clinical significance
Skin	Ideal skin is not too thick, not too thin, and has minimal sebaceous glands	<ul style="list-style-type: none"> Excessively thick skin will obscure surgical corrections and increase risk of polly-beak deformity Thin skin exposes contour irregularities
Superficial muscular aponeurotic system (SMAS)	Nasal muscles are enveloped by SMAS just as other muscles of facial expression	When dissection is performed in a sub-SMAS plane, open rhinoplasty is relatively bloodless
Nasal bones	Paired bones project from frontal bone and overlie the upper lateral cartilages; ensures a smooth transition along brow-tip esthetic line from medial brow to nasal tip	Trauma can disrupt this relationship, resulting in an irregular fronto-nasal-orbital contour between the upper and middle nasal thirds
Upper lateral cartilages	<ul style="list-style-type: none"> Join septum medially to form (internal) nasal valves and join the nasal bones cephalically to form keystone area Normal nasal valve is the narrowest area of the airway with an angle of 10 to 15 degrees; this angle can be widened with a spreader graft 	<p>Crooked noses are either deviated or twisted</p> <ul style="list-style-type: none"> Deviation: Nasal bones and upper lateral cartilages both point away from the midline Twisted: Nasal bones and upper lateral cartilages point in opposite directions
Lower lateral cartilages	<ul style="list-style-type: none"> Cephalic margins of the lower lateral cartilages usually overlie, or are sometimes continuous with, the upper lateral cartilages; this area is called the <i>scroll</i> Lower lateral cartilages can be divided into medial, intermediate, and lateral crura; intermediate crus can be further subdivided into the lobular and domal segments Most prominent point of the dome is the tip-defining point 	<ul style="list-style-type: none"> Cephalic trim of the lower lateral cartilage decreases tip bulbousness Resiliency of the lower lateral cartilage determines the amount one can safely remove Sufficient width (typically ~6 mm) must be preserved to prevent postoperative external nasal valve collapse
Major tip supports	<ol style="list-style-type: none"> Direction, strength, and resiliency of lower lateral cartilage Attachment of medial crura to inferior septal angle Attachment of lower and upper lateral cartilage 	Nasal tripod model: Predicts changes in tip position based on changes to the legs of the tripod (conjoined medial crura and two lateral crura)

(Nasal structures cont)

	Description	Clinical significance
Minor tip supports	<ol style="list-style-type: none">1. Attachment of soft tissue envelope to alar cartilages2. Anterior nasal spine3. Membranous septum4. Cartilaginous septal dorsum5. Sesamoid complex6. Interdomal ligament between lower lateral cartilages	<ul style="list-style-type: none">• The lower lateral cartilages are divided (dome division) by sectioning the interdomal ligament• Intercartilage sutures reconnect the divided lateral cartilages, and intracartilage sutures reshape each lateral cartilage individually
Tip rotation	<ul style="list-style-type: none">• Determined by columellar-labial/nasolabial angle; normal values are 90 to 105 degrees (males) and 95 to 110 degrees (females)• Caudal septal excess can create a wide nasolabial angle and a short upper lip; the tip will appear over-rotated	<ul style="list-style-type: none">• To increase tip rotation: Remove hump, cephalic trim or shorten lateral crura, augment medial crura, or premaxillary shield graft• To decrease tip rotation: Caudal septum excision, shorten medial crura, augment dorsum
Tip projection	<ul style="list-style-type: none">• Determined by the distance between the nasal tip and the facial plane• Rough estimate is a 3:4:5 triangle (columella:base:dorsum)• Goode method: Tip projection should be 0.55 to 0.6 of nasal length• Simon method: Upper lip length = nasal projection (lip to tip ratio = 1:1)• Basal view should reveal an isosceles triangle with a columella to lobule ratio of 2:1	<ul style="list-style-type: none">• To increase tip projection: Transdomal or interdomal sutures, shield graft, columellar strut graft• To decrease tip projection: Complete transfixion incision, shorten crura (excise strip of lateral and/or medial crura and then reattach), lower septal angle• Don't forget that underprojected chin accentuates an overprojected nose
Septum	<ul style="list-style-type: none">• Membranous septum (fibrofatty connective tissue caudal to cartilaginous septum) versus cartilaginous septum (quadrangular cartilage) versus bony septum (perpendicular plate of ethmoid, vomer)• Vomer sits on maxillary crest	<ul style="list-style-type: none">• Sufficient cartilage must be maintained dorsally and caudally (typically 1-cm L-strut) during septoplasty to avoid tip ptosis• Approaches to septum: Closed (Killian, hemi-, partial, and complete transfixion), open (external rhinoplasty and Le Fort I)
Inferior turbinate	<ul style="list-style-type: none">• Most common source of nasal obstruction• Hypertrophy is either bony or mucosal	<ul style="list-style-type: none">• Nonsurgical: Antihistamines, decongestants, topical steroids• Surgical: Turbinoplasty (submucosal resection, radiofrequency ablation)

Surface anatomy*

	Description	Clinical significance
Brow-tip esthetic line	Line connecting the brow to the nasal tip should be smooth, symmetric, unbroken	Brow shape/position as well as dorsal irregularities affect this line
Soft tissue glabella	Most prominent point of forehead between brows	Tangential lines from soft tissue glabella and the nasal dorsum form the nasofrontal angle
Radix	<ul style="list-style-type: none">• Junction of the frontal bone and the nasal dorsum• Normal vertical position approximates the supratarsal crease• Normal projection is 4 to 9 mm anterior to cornea• Serves as the apex of the nasofrontal angle (normally ~120 degrees)	Low radix disproportion gives the illusion of an overprojected nasal tip and a pseudo-hump; this is corrected by augmenting the radix
Rhinion	<ul style="list-style-type: none">• Junction of bony and cartilaginous dorsum• At rhinion the upper lateral cartilage and septum are continuous, whereas they are connected by a fibrous union near the septal angle	Osteotomies can collapse this region, resulting in nasal obstruction, especially if nasal bones are short
Nasal height and width	Roughly, height should = $\frac{1}{3}$ facial height and width should = $\frac{1}{5}$ facial width	Actual nasal length (distance from radix to tip) should be 0.67 times middle face height
Tip-defining points	<ul style="list-style-type: none">• Light reflects from the nasal tip at four points: Centrally a supratip break (junction of the nasal dorsum and lobule); at infratip break (junction of the lobule and columella); and laterally at the most projected area of each lower lateral cartilage• These lateral points normally are 5 to 6 mm apart	If tip-defining points are abnormally positioned, dome division with subsequent intra- and interdomal sutures may be necessary
Sill	The floor of the nasal opening formed by the continuation of the ala and the columellar base	Nostril sill excision narrows the alar base, whereas alar lobule flaring is treated with crescent excisions along alar-facial groove
Ala	<ul style="list-style-type: none">• Alar rim should be 2 to 3 mm cephalic to columella (normal columellar show)• Ala can be hanging, normal, or retracted	Alar retraction can occur if inadequate cartilage remains after cephalic trim of lower lateral cartilage

*Dorsal length, tip projection, and alar base width vary with ethnic norms and esthetic preferences.

Blood supply

External carotid artery	External nose	Internal nose
Facial artery	Angular, lateral nasal, alar, septal, superior labial arteries	N/A
Maxillary artery	N/A	Sphenopalatine (supplies posterior septum, lateral wall, roof and floor), greater palatine (supplies anterior/inferior septum)
Internal carotid artery	External nose	Internal nose
Ophthalmic artery	Dorsal nasal artery	Anterior ethmoid (supplies anterior/superior septum and lateral nasal wall), posterior ethmoid (supplies septum, lateral nasal wall, superior turbinate)
Anterior ethmoid artery	External nasal artery	N/A
Kiesselbach plexus (Little area)	<ul style="list-style-type: none">• Confluence of arteries at anterior septum; contributions from internal (anterior ethmoid) and external (sphenopalatine, greater palatine, and superior labial) carotid arteries• Common site for anterior epistaxis	<ul style="list-style-type: none">• Woodruff plexus = venous plexus on lateral nasal wall with contributions from internal maxillary, posterior nasal, ascending pharyngeal, sphenopalatine arteries• Common site for posterior epistaxis

Rhinoplasty Techniques

Incisions	Location	Notes
Marginal	<ul style="list-style-type: none">• Caudal margin of lower lateral cartilage• Combine with transcolumellar incision for open/external rhinoplasty	While open rhinoplasty preserves all of the major tip supports, it weakens tip support more dramatically than endonasal surgery
Intercartilaginous	Cephalic margin of lower lateral cartilage/caudal margin of upper lateral cartilage (scroll)	Sacrifices one of the major tip supports during closed/endonasal rhinoplasty
Intracartilaginous (cartilage splitting)	1 mm above caudal margin of upper lateral cartilage	Provides access to nasal dorsum and, if combined with marginal incisions, allows retro-delivery of lower lateral cartilages
Transcolumellar	An inverted “V” placed in the most narrow portion of the columella	Blood supply to tip derived from lateral nasal artery so transcolumellar incision does not endanger tip

(Rhinoplasty Techniques cont)

Incisions	Location		Notes
Septal	One or both sides of septal mucosa	Disarticulation of medial crural footplates from septum	<ul style="list-style-type: none">• Hemi (incomplete), partial, and complete transfixions are all placed at the caudal border of cartilaginous septum; Killian incision is placed 1 to 2 cm posterior to caudal border• Less rents with transfixion incisions (made within squamous epithelium) than Killian incisions (made within respiratory epithelium)• Transfixation incision completely cuts across the fixation
Complete transfixion	Both sides	Yes	
Partial transfixion (incomplete)	Both sides	No	
Hemitransfixion	One side	Yes	
Killian	One side	No	
Osteotomy	<ul style="list-style-type: none">• Medial osteotomies either parallel midline (to correct deviated nasal bones) or fade laterally (used with straight nasal bones)• Lateral osteotomies are made either low or high (relative to starting point on piriform rim)		Used to close open-roof deformities after hump removal, straighten crooked noses, and flatten convex nasal bones

Rhinoplasty Complications

	Description	Treatment (10% to 15% of rhinoplasties require revision)
Infection	<ul style="list-style-type: none">• Rare (< 2% risk); includes cellulitis, abscess, and granuloma• Higher incidence associated with grafting, especially when using alloplasts• Toxic shock, mediated by <i>Staphylococcus aureus</i>, occurs in 0.016% of cases	<ul style="list-style-type: none">• Appropriate antibiotics +/- incision and drainage may be indicated• In the setting of toxic shock: Remove and culture nasal packs, begin empiric beta-lactamase resistant antibiotic therapy, provide hemodynamic resuscitation, and investigate multisystem failure
Skin necrosis	May be caused by overly thinned soft tissue flaps, excessive undermining, electrocautery, dorsal onlay grafts and external nasal splints	<ul style="list-style-type: none">• Initial conservative management: Serial debridement allowing healing via secondary intention• Local flap reconstruction, if needed, should respect nasal esthetic subunits
Polly-beak deformity	<ul style="list-style-type: none">• Convex nasal dorsum (at caudal ⅔) resulting in supratip prominence/fullness• Causes include excess resection of bony dorsum, inadequate resection of cartilaginous dorsum, loss of tip support, shortened columella, scarring	<ul style="list-style-type: none">• Nonsurgical: Massage, steroid injections• Surgical: Augment upper ⅓ of dorsum, excise additional cartilaginous dorsum, columellar strut graft

(Rhinoplasty Complications cont)

	Description	Treatment (10% to 15% of rhinoplasties require revision)
Rocker deformity	<ul style="list-style-type: none"> • Caused by osteotomies that are placed too far cephalically (approaching or beyond the frontonasal junction) • Cephalic aspect of fractured bones is lateralized when attempt made to narrow caudal bony complex • Inadvertent “rocking” (in the coronal plane) results as the cephalic portion of the bony complex hinges on a fulcrum near the radix 	A stab incision near the radix provides transcutaneous access for a 2-mm osteotome to re-create a fracture at a more appropriate caudal position
Saddle-nose deformity	<ul style="list-style-type: none"> • Concave nasal dorsum • Causes include excess resection of dorsum and open roof deformity 	<ul style="list-style-type: none"> • Lateral osteotomies (close the open roof) if mild • Dorsal graft if more severe
Nasal obstruction	<ul style="list-style-type: none"> • Long-term postoperative obstruction: Persistent septal deformity, narrowing of internal nasal valve, external nasal valve collapse, and synechia • Short-term postoperative obstruction: Expected due to edema 	<ul style="list-style-type: none"> • Revision septoplasty required to correct persistent septal deformity • Spreader grafts correct narrowed nasal valve • Batten grafts correct external nasal valve collapse • Nonsurgical synechia treatment: Steroid injections • Surgical: Lysis (+/- rotational mucosal flaps) and splinting
Retracted ala	<ul style="list-style-type: none"> • Excess cephalic trim and/or inappropriate marginal incision closure can cause alar retraction and excess columellar display • On frontal view, retracted ala replace the gentle curves of the esthetic alar-columellar relationship described as a “gull in flight” with harsh angles 	<ul style="list-style-type: none"> • A narrow strip of auricular or lower lateral cartilage (harvested via cephalic trim) is placed in a pocket just caudal to a marginal incision • A composite graft (skin and cartilage) may be used if vestibular skin requires augmentation
Nasal tip	<ul style="list-style-type: none"> • Over-rotation and ptosis usually result from inappropriate preoperative planning • Bossae (unsightly protuberances) are caused by a buckling of the lower lateral cartilage during skin contraction; patients with thin skin, strong cartilage, and bifid tips are at risk 	<ul style="list-style-type: none"> • See tip rotation and tip projection in the Nasal Structures table on page 403 • Bossae may be corrected via marginal incisions allowing domal resuturing or cartilage trimming +/- camouflage onlay grafts (cartilage or fascia)
Septal perforation	<ul style="list-style-type: none"> • Results from inadequate subperichondrial dissection, with increased risk at sites of previous fracture or surgery, septal spurs, and marked deviation • Perforations cause turbulent airflow resulting in dry, bleeding/crusting mucosa (and whistling if located anteriorly) • Chronic infection, tip ptosis and nasal saddling are potential late sequelae 	<ul style="list-style-type: none"> • Surgical closure is recommended for symptomatic perforations • Local intranasal flaps, usually from nasal floor, are more successful when accompanied with interpositional grafts (crushed cartilage, temporalis fascia, acellular human dermis) • Silicone septal buttons are an alternative option to surgery

Otoplasty Anatomy (Fig 12-4)

	Description	Clinical significance
Surface anatomy	<ul style="list-style-type: none">• Auriculocephalic angle (20 to 30 degrees) = angle the helix projects from the skull<ul style="list-style-type: none">– Determined by the conchomastoid angle (normal = 90 degrees), the conchoscaphal angle (normal = 90 degrees), and the length of the cartilage• Normal auricular projection on frontal view is 2 to 2.5 cm• Normal ear height is 6 cm	<ul style="list-style-type: none">• Increasing either the conchoscaphal or the conchomastoid angles will result in prominauris (auriculocephalic angle > 30 degrees)• Unfurled helix is most common ear abnormality that results in prominauris because of an obtuse conchoscaphal angle (up to 150 degrees)• Concha hypertrophy causes prominauris because of an obtuse conchomastoid angle
Prominauris (protruding ear)	Three causes: Conchal hypertrophy, lack of antihelical fold, combination of the two	Correct prominauris with conchal setback, creation of antihelical fold, or a combination of the two

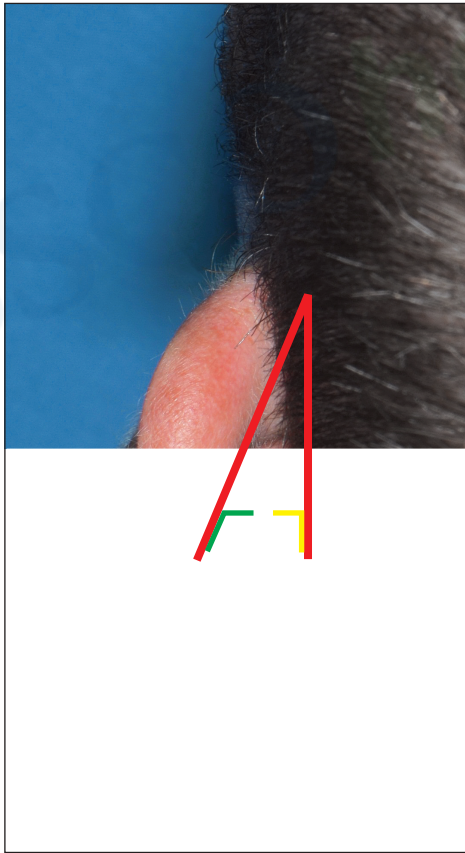


Fig 12-4 Surface anatomy of the ear. Conchomastoid angle (*yellow*, normal = 90 degrees). Conchoscaphal angle (*green*, normal = 90 degrees). Auriculocephalic angle (*red*, normal = 20 to 30 degrees).

Blood supply

Superficial temporal artery	Runs 1 cm anterior to ear, deep to anterior auricular muscle
Posterior auricular artery	Runs posterior to posterior auricular crease deep to both posterior auricular muscle and greater auricular nerve

Nerves

Trigeminal (auriculotemporal branch)	Helical crus, tragus
Greater auricular	Entire anterior auricle except tragus and superomedial aspects (auriculotemporal)
Lesser occipital	Posterior auricular surface
Vagus (Arnold nerve)	Posterior aspect of external auditory meatus and lateral aspect of tympanic membrane
Glossopharyngeal (Jacobson nerve)	Medial aspect of tympanic membrane

Otoplasty Techniques

	Description	Clinical significance
Creation of antihelical fold	<ul style="list-style-type: none">• Mustardé technique creates antihelical fold via 3 to 4 horizontal mattress sutures along the scapha• Some advocate cartilage modification techniques in addition to sutures to lessen relapse• Helix should project 2 to 5 mm lateral to antihelix on frontal view	<ul style="list-style-type: none">• Minimizes auricle protrusion by reducing the auriculocephalic and conchoscaphal angles• Reverse telephone ear deformity results from overcorrection of antihelical fold (and/or overcorrection of protruding lobule)
Conchal setback	<ul style="list-style-type: none">• Concha sutured to mastoid periosteum +/- cartilage modification (resecting cartilage from concha cavum)• Furnas technique relies on sutures alone and has a higher rate of relapse	<ul style="list-style-type: none">• Minimizes auricle protrusion by reducing the auriculocephalic and conchomastoid angles• External auditory meatus narrowing results if concha is sutured anteriorly• Furnas = failure

Otoplasty Complications

	Description	Treatment
Hematoma	<ul style="list-style-type: none"> Rare (3% incidence), usually heralded by unilateral pain Hematoma can lead to infection and/or cartilage necrosis or cauliflower ear deformity (unevacuated hematoma consolidates, becoming cartilaginous) 	<ul style="list-style-type: none"> Prevent with prophylactic drains Aspiration rarely adequate Surgical drainage consists of suture release, evacuation, and reapplication of pressure dressing
Infection	<ul style="list-style-type: none"> Perichondritis can lead to suppurative chondritis, which can lead to cartilage necrosis <i>Pseudomonas aeruginosa</i>, <i>Staphylococcus aureus</i>, and <i>Escherichia coli</i> are the usual culprits 	<ul style="list-style-type: none"> Incision and drainage necessitates removal of all sutures Serial debridements may be indicated if necrotic cartilage is present
Telephone ear deformity	<ul style="list-style-type: none"> Middle third of the auricle is corrected/overcorrected with respect to upper and lower thirds Typically occurs after conchal bowl setback without addressing antihelical and lobule deformities 	<ul style="list-style-type: none"> Address antihelical fold deformities (Mustardé), and lobule deformities, if present Contrast with reverse telephone ear deformity (middle third projects more than upper and lower thirds after antihelical fold creation and/or protruding lobule correction)
Keloid formation	<ul style="list-style-type: none"> Rare (~2% incidence) While both keloids and hypertrophic scars are elevated, only keloids overgrow the original wound boundary Most likely site is postauricular incision; tension-free closure is paramount 	Triamcinolone injections, excision (either within or beyond the keloid margin), pressure dressings, silicone sheeting, and low-dose radiation have all been used
Relapse	<ul style="list-style-type: none"> Loss of correction occurs up to 40% with suture-only techniques Inadequate suture bites, cartilage tearing, and cartilage resilience increase relapse 	Cartilage weakening and/or modification techniques decrease recurrence

Lower Third Esthetic Surgery

- Superior border: Subnasale (point where upper lip becomes columella)
- Inferior border: Menton (most inferior point on soft tissue chin)

Facelift Anatomy

	Description	Clinical significance
Skin	Ideal facelift candidates: 40s and 50s without a history of significant sun exposure, smoking, recent weight loss, hypertrophic scar/keloid formation, or collagen vascular disease	<ul style="list-style-type: none">• Ultraviolet-induced actinic changes result in skin laxity that can worsen cervicomen- tal definition and jowling• The extent of skin redundancy directly cor- relates with the degree of skin undermining required• Smokers have 12× increased risk of skin slough• Less undermining = preservation of more vas- cular perforators to skin = less skin slough
Subcutaneous fat	Maintain 4 to 5 mm of subcutaneous fat attached to skin flap to preserve adequate blood supply	Areas of subcutaneous liposuction are patient specific but typically liposuction is not per- formed in the preauricular and postauricular areas
Platysma	Medial borders have a variable degree of decussation across midline and may become redundant with age (platysmal banding)	Platysmaplasty treats platysmal banding by midline approximation of the medial borders via corset suture (+/- myotomy)
SMAS	<ul style="list-style-type: none">• Contractile fibromuscular fascia that envelops muscles of facial expression• Contiguous with the platysma below the mandible and the temporopari- etal fascia above the zygomatic arch	<ul style="list-style-type: none">• Dermal connections between SMAS and facial skin centrally result in improved skin contours during repositioning of the SMAS• Some discourage undermining the melola- bial fold to preserve the integrity of these connections
Retaining ligaments of the face	<ul style="list-style-type: none">• Facial skin is anchored by these liga- ments at various points on the face• The two osseocutaneous ligaments are the zygomatic (located just pos- teroinferior to malar eminence) and mandibular (located along jawline in the parasymphiseal region)• The two fasciocutaneous ligaments are the parotid and masseteric	<ul style="list-style-type: none">• Ptotic soft tissues hang off the face except in the parasymphiseal region where tissues tightly adhere by the mandibulosseocutane- ous ligament• The juxtaposition of these unsupported ptotic tissues and the adjacent supported tissues serve as the anatomical basis of jowling and the formation of the prejowl sulcus/crease• Release of the retaining ligaments allows repositioning of the tissues during a deep plane facelift

Nerves

	Description	Clinical significance
Facial nerve	<ul style="list-style-type: none"> • Five branches and corresponding muscles of innervation include: temporal-frontalis, upper orbicularis oculi, corrugator supercilii, procerus, zygomatic, lower orbicularis oculi, corrugator supercilii, buccal zygomaticus major and minor, levator anguli oris, buccinator, upper orbicularis oris, mandibular lower orbicularis oris, depressor anguli oris, depressor labii inferioris, mentalis, cervical platysma • Innervation of these muscles is from the deep surface except the buccinator, levator anguli oris, and mentalis 	<ul style="list-style-type: none"> • Facial nerve runs deep to superficial in a proximal to distal fashion • It courses proximally within the parotid, dividing the parotid into superficial and deep lobes; this ensures safe subSMAS dissection proximally • Distally the facial nerve becomes superficial, innervating the anteromedial muscles of facial expression from the superficial aspect; this prevents safe subSMAS dissection at the labial commissure
Temporal branch of cranial nerve VII	<ul style="list-style-type: none"> • Crosses zygomatic arch ~2 cm anterior to external auditory meatus • Overlying the arch, the temporal branch runs just deep to the subcutaneous tissue and a thin layer of SMAS 	<ul style="list-style-type: none"> • Dissection toward the lateral canthal region must be in subcutaneous tissue or subperiosteal plane to preserve temporal branch • Temporal branch is the most common facial nerve branch injured during facelift
Marginal mandibular branch of cranial nerve VII	Crosses mandibular margin up to 1.5 cm anterior to the facial artery	Greatest risk of damage occurs during subplatysmal dissection caudal to the mandible
Great auricular nerve (GAN)	Exits posterior border of sternocleidomastoid 6 to 7 cm caudal to ear canal (Erb point/punctum nervosum)	<ul style="list-style-type: none"> • Most common nerve injured during facelift • Skin flap elevation at Erb point places GAN at risk as little or no platysma overlying sternocleidomastoid laterally

Facelift Terminology

	Description	Clinical significance
Melolabial crease (nasolabial fold)	<ul style="list-style-type: none"> The crease results from the insertion of the SMAS and the muscles originating from the zygoma Loss of malar support results in malar fat pad descent, deepening this crease 	<ul style="list-style-type: none"> Facelifts address prominent nasolabial folds during SMAS resuspension Temporal extensions and deep plane techniques ensure fold elimination
Cervicomental definition/chin-throat angle	Angle formed by horizontal plane of the submental region and the vertical plane of the neck; normal 90 degrees	Causes of obtuse cervicomental angle include relaxed skin, excess fat, lax platysma (Turkey gobbler deformity), retrognathia/microgenia, and a low hyoid (see Dedo classification on page 393)
McGregor patch	<ul style="list-style-type: none"> Also known as <i>zygomatic ligament</i> Facial and transverse facial arteries, along with zygomatic branch of facial nerve, are found in this region 	Surgeons must be cautious operating in this region as damage to arteries and nerves can easily occur
Short versus long flap	Skin flap length is determined by the amount of subcutaneous tissue undermined	Short flap dissection ~5 cm radially around ear, whereas long flap subcutaneous tissue undermining is > 5 cm
SMAS manipulation	<ul style="list-style-type: none"> SMAS plication: Uncut SMAS is sutured on itself (SMAS not incised); plicate = fold, as in fundoplication SMAS imbrication: SMAS is incised but not excised (a SMAS flap is raised, redraped posteriorly, and sutured on top of the immobile SMAS); imbricate = "imbricat" SMASectomy: Incise and excise a portion of SMAS before resuspending. There is no SMAS overlap after a SMASectomy (both SMAS plication and SMAS imbrication result in SMAS overlap) 	<ul style="list-style-type: none"> Plication less risky but preserved redundant SMAS can look bulky in preauricular region (bad in heavy patients) Imbrication more risky because parotid capsule may be unintentionally incised (may lead to cranial nerve VII damage or postoperative sialocele), but redundant SMAS addressed (good in heavy patients)
Tumescent versus nontumescent techniques	Injection of a large volume of dilute lidocaine and epinephrine into subcutaneous fat (see page 416)	Tumescence can provide hydrodissection and facilitate hemostasis; performed +/- liposuction
Preauricular incision (male versus female)	Men (and women with hirsute preauricular skin) will not tolerate an endaural preauricular incision, whereas nonhirsute women will	Incisions made in the preauricular sulcus will preserve/maintain nonbearded skin even after bearded skin is advanced posteriorly; an endaural incision in a man would replace bearded preauricular skin with nonbearded skin

Facelift Techniques

	Description	Advantages	Disadvantages
Skin only	<ul style="list-style-type: none"> • Development and redraping of a lipocutaneous flap (skin and ~5 mm subcutaneous fat) • Not routinely performed 	<ul style="list-style-type: none"> • Minimal surgical time • Minimal risk of nerve damage 	<ul style="list-style-type: none"> • Short-lived result • Unesthetic, wind-blown appearance
Skin with SMAS manipulation	Development of a lipocutaneous flap with manipulation of the underlying SMAS	<ul style="list-style-type: none"> • Allows for SMAS plication • Less technique sensitive, minimal surgical time, low facial nerve risk 	<ul style="list-style-type: none"> • Less dramatic outcome • Plication can cause redundancy/bunching in preauricular region
Biplane	Creates two flaps (lipocutaneous and SMAS)	<ul style="list-style-type: none"> • Less tension on skin flap • More natural appearance (less bunching of tissue) 	More technically sensitive than SMAS plication because elevating SMAS off parotid fascia increases facial nerve injury risk
Deep plane	Often one thick flap (skin, subcutaneous fat, SMAS/platysma) extending medial to the zygomaticus muscles	<ul style="list-style-type: none"> • Enhanced midface improvement • Better blood supply to, and greater tensile strength of, the flap 	<ul style="list-style-type: none"> • Technically sensitive • Longer operating time • Higher risk for nerve injury • Prolonged postoperative edema/recovery
Composite	Similar to deep-plane facelift but includes addition of orbicularis oculi	Addresses periorbital aging due to inferiolateral descent of the orbicularis oculi	Requires lower blepharoplasty skin incision
Mini-facelift	<ul style="list-style-type: none"> • “Short scar/minimal incision” • Numerous incision techniques exist but all exclude postauricular extension 	Least operative time, least invasive (enabling poor healers to be potential surgical candidates), rapid recovery	Result less pronounced and shorter lasting
Subperiosteal	Corrects ptotic soft tissues of the upper facial two-thirds; access to upper third via endoscopic versus open approach, while midface access provided by intraoral versus subciliary incision	<ul style="list-style-type: none"> • Provides superior displacement of the facial muscles • Excellent midface correction • Excellent blood supply to the undermined tissues 	<ul style="list-style-type: none"> • Requires separate transoral, transtemporal, and transorbital incisions • Longer postoperative edema

Facelift Complications

	Description	Treatment
Bleeding and hematoma (5% incidence)	<ul style="list-style-type: none"> Common sources are subcutaneous tissue, McGregor patch, and external jugular vein Generally bipolar cautery preferred over monopolar, and ligation preferred over bipolar cautery near facial nerve branches Tumescence improves intraoperative hemostasis Intraoperative and postoperative blood pressure control are essential as blood pressure control is the most crucial preventative measure 	<ul style="list-style-type: none"> Evacuation Minor hematomas evacuated via “milking” or aspirating (repeatedly as needed) Major hematomas evacuated via release of skin sutures, visualization of source, cauterization versus ligation Pressure dressings must not inhibit flap perfusion Untreated hematomas can lead to chronic skin irregularity, fibrosis, and discoloration
Skin slough and necrosis (1% to 3% incidence)	<ul style="list-style-type: none"> Causes include excess skin tension, overly thin skin flap, and hematoma Smokers have 12× increased risk Long flaps also increase risk 	<ul style="list-style-type: none"> Conservative wound care until eventual epithelialization Most common site is postauricular region
Nerve injury	<ul style="list-style-type: none"> Sensory nerves at risk include terminal cutaneous branches of the face/neck and the great auricular nerve (most common sensory nerve injury; occurs in 1% to 7% of patients) Motor nerve injury occurs ~2% of patients Literature cites both temporal and marginal branches of cranial nerve VII as the most commonly injured branches; the technique used determines which branch is at greatest risk 	<ul style="list-style-type: none"> Majority (85%) of motor nerve injuries resolve without treatment; these are likely to occur secondary to traction or cautery Skin sensation typically returns within 6 to 8 weeks unless major nerve injured Observed transection of the greater auricular nerve or a branch of cranial nerve VII necessitates immediate microsurgical repair
Earlobe deformity	Excess tension placed on the flap adjacent to the earlobe leads to caudal contraction (result called <i>pixie</i> , <i>elfin</i> , or <i>satyr ear deformity</i>)	Correct with VY closure 6 months postoperatively, or do revision facelift if vertical scar from VY closure unacceptable
Hairline changes	Poor incision placement may lead to loss/displacement of temporal tuft, alopecia (excess tension), and stair-stepped hairline at postauricular flap	<ul style="list-style-type: none"> Wait 3 to 6 months for resolution of hair loss (telogen effluvium) If alopecia persists, primary excision, rotational flaps, or micrografting may be indicated
Parotid injury	Violation of parotid capsule can result in sialocele or fistula formation	<ul style="list-style-type: none"> Intraoperative: Suture capsule closed and then oversew with SMAS Postoperative: Needle aspiration, pressure dressing If persists, liquid diet, antisialogogues, and drains may be indicated
Local anesthesia toxicity	See liposuction complications on page 417	See liposuction complications on page 417

Liposuction and Autologous Fat Transfer Techniques

- Liposuction is surgical aspiration of subcutaneous adipose tissue using negative pressure suction
- Lipomatosis is abnormal localized accumulation of fat (also a tumorlike accumulation of fat)

	Description	Clinical significance
Dry technique	No tumescence used prior to suctioning of fat	Infrequently used; bleeding complications are common
Tumescent technique	<ul style="list-style-type: none"> • Wet technique; infiltrate large volumes of wetting solution via cannula through stab incision until tissue turgor (“peau d’orange”) is achieved • Wetting solution formula example: 1 L normal saline, 1 mg epinephrine (1 amp of 1:1,000), 50 mL 1% idocaine +/- bicarbonate • Submental stab incision +/- retrolobular stab incisions facilitate submental, neck, and jowl liposuction 	<ul style="list-style-type: none"> • 2:1 to 3:1 is the ratio of tumescent to lipoaspirate volume • Approximately 70% of fluid injected will be absorbed • Lidocaine (0.05% to 0.2%) and epinephrine (1:500,000 to 1:1,000,000) decreases postoperative bruising and pain • Allow 7 to 10 minutes for vasoconstriction • Can be performed in isolation or in combination with cervicofacial rhytidectomy
Lipodystrophy	A disturbance of fat metabolism typically characterized by progressive and symmetric loss of subcutaneous fat	Often treated with autologous fat transfer
Autologous fat transfer	<ul style="list-style-type: none"> • Larger cannula diameters cause less fat damage during harvest • Harvest sites include abdomen and medial or lateral thigh • Processing fat typically includes centrifugation to separate into supranatant (triglycerides, oil), middle fat layer, and infranatant (fluid, blood) 	<ul style="list-style-type: none"> • Avoid central necrosis at recipient site by layering smaller aliquots • Common recipient sites include the nasojugal trough, melolabial fold, and lips

Liposuction Complications

Lidocaine toxicity	<ul style="list-style-type: none"> • 7 mg/kg (or manufacturer's recommendation) safest for cervicofacial liposuction; subcutaneous lidocaine doses are generally safe up to 35 mg/kg although limit for cervicofacial toxicity is less than body toxicity • Central nervous system (CNS) affected before cardiovascular system (CVS) <ul style="list-style-type: none"> – CNS effect: Depressed cortical inhibitory pathways lead to unopposed excitatory pathways – CVS toxicity: Direct myocardial depression, and cardiac conductance and vascular smooth muscle depressant; drop in blood pressure, increased or decreased heart rate, ventricular fibrillation, cardiac arrest • Early signs (3 to 6 µg/mL [plasma]): Light-headedness, restlessness, tinnitus, slurred speech, metallic taste, perioral numbness • Later signs (6 to 9 µg/mL [plasma]): Shivering, muscle twitches, tremors • Late signs (>10 µg/mL [plasma]): Convulsions, loss of consciousness, grand mal seizures, apnea, cardiovascular collapse • Treatment <ul style="list-style-type: none"> – To terminate seizure, hyperventilate patient with 100% oxygen (consider intubation if full stomach); if needed, intravenous diazepam 0.1 mg/kg – Intravenous fluids +/- phenylephrine, if hypotensive – Advanced cardiovascular life support (ACLS) protocol for arrhythmias
Infection	<ul style="list-style-type: none"> • Rare (0.2% cases) • Usually secondary to hematoma
Hematoma, seroma	<ul style="list-style-type: none"> • Avoid by using postoperative compression dressing at surgical site • Treatment: Needle aspiration and pressure dressing
Contour irregularity	<ul style="list-style-type: none"> • Due to under/overcorrection and abnormal skin retraction • Treatment: Removal of more fat, fat injection, or skin tightening/excision • Cobra deformity is a neck concavity that results from over-resection of submental subcutaneous (and more likely subplatysmal) fat
Skin flap dermis healing to platysma	<ul style="list-style-type: none"> • If cannula aperture is directed toward the skin during liposuction, the skin flap becomes overly thinned and leads to unesthetic outcome as dermis heals to platysma
Nerve injury	<ul style="list-style-type: none"> • Involvement of greater auricular or facial nerve branches • Avoid by instrumenting only within preoperative markings

Ancillary Procedures

Skin Rejuvenation

Pretreat skin for 6 weeks (keratinocyte maturation from stratum basale to stratum corneum takes 6 weeks) prior to chemical peels or laser resurfacing:

- 4% hydroquinone two times a day: Decreases risk of hyperpigmentation; inhibits melanocyte activity (inhibits tyrosinase—less conversion of tyrosine to melanin = less melanin)
- 0.05% to 0.1% tretinoin at bed time: Increases re-epithelialization via increased fibroblast production of collagen; also increases penetration of hydroquinone (and subsequent chemical peels, if used)
 - Remember: Tretinoin = retinoic acid (Retin-A) while isotretinoin = accutane and is contraindicated for 1 year; think “I-SO”-tretinoin like “EYE-SORE”-tretinoin)
- Sunblock every morning: Sun protection factor (SPF) ≥ 30
- Possible antiviral therapy starting one day prior to and continued 10 to 14 days after procedure completed; recommended for patients with a history of viral infections
- Antibiotic +/- antifungal therapy starting 1 day prior to and continued after procedure

Chemical Peel

	Description	Clinical relevance
Epidermis	<ul style="list-style-type: none">• 0.06 mm thick• Composed of five layers (superficial to deep): Stratum corneum, lucidum, granulosum, spinosum, basale (Californians Like Green String Bikinis)• Very superficial peels remove stratum corneum only, whereas superficial peels can penetrate to epidermis-dermis junction	<ul style="list-style-type: none">• Superficial peels (Jessner, 20% to 35% glycolic acid, 15% to 50% salicylic acid, 10% to 20% trichloroacetic acid [TCA]) penetrate ~0.06 mm• Used to mitigate mild photoaging, treat comedonal acne, and inflammatory erythema• Repeated superficial peel \neq medium depth peel
Dermis	<ul style="list-style-type: none">• Composed of two layers (superficial to deep): Papillary (0.45 mm), and reticular (0.6mm)• Healing occurs from epithelium at wound margins and adnexal structures within defect; fewer adnexal structures = greater risk of scarring• Deep reticular dermis may heal by re-epithelialization but risks scarring• Full-thickness insults cannot heal by re-epithelialization and will scar	<ul style="list-style-type: none">• Medium depth peels (35% TCA): Penetrates papillary dermis and treats mild to moderate photoaging (fine wrinkles, actinic changes, dyschromias)• Deep peels (Baker-Gordon phenol): Penetrates upper reticular dermis; treats severe photoaging (deep rhytids)• By comparison, a split-thickness skin graft is typically 0.38 mm thick

Laser Resurfacing

- Lasers are continuous or pulsed and either ablative (targets epidermis and upper papillary dermis) or nonablative (targets dermis and spares epidermis)
- Chromophore for carbon dioxide (CO₂) laser = water
- Fractional CO₂ lasers ablate the majority but not all tissue within the spot size; the numerous minuscule spared areas serve as foci for re-epithelialization and speed recovery

Fillers

- Injectable: Tissue derived (eg, Zyderm, Zyplast [Allergan]) require skin testing; synthetic (eg, Juvederm [Allergan]; Restylane [Galderma]) are hyaluronic acid derivatives that do not require skin testing
- Implantable: Tissue-derived (Alloderm [LifeCell]) versus synthetic (silicone, rapidly expanded polytetrafluoroethylene [ePTFE])
- Autologous tissue transfer: Injectable (fat) or implantable (dermal fat grafts)

Botulinum Toxin

- Endotoxin produced by *Clostridium botulinum* (anaerobic Gram-positive rod)
- Seven serotypes (botulinum toxin A to G) inhibit exocytosis of acetylcholine at neuromuscular junction (botulinum toxin A targets SNAP-25)
- Used to correct horizontal forehead, vertical glabellar, lateral orbital, radial rhytids

Muscle	Findings	Site and dose
Frontalis	Horizontal/transverse forehead lines	<ul style="list-style-type: none"> 10 to 25 units (5 to 10 sites on brow/forehead) Stay 1 cm away from orbital rim, often inject in V pattern to preserve some inferolateral frontalis function ("chemical brow lift")
Procerus	Horizontal/transverse glabellar lines (scowl lines)	2.5 to 5 units (1 to 2 sites on central glabella)
Corrugator	Vertical glabellar lines ("11s")	2.5 to 5 units (1 to 2 sites per corrugator)
Transverse nasalis	Radial lines along nasal dorsal side wall (bunny lines)	2.5 to 5 units (1 site per side wall)
Orbicularis oculi	Lateral/radial orbital lines	<ul style="list-style-type: none"> 7.5 to 15 units per side Stay 1 cm lateral to orbital rim
Platysma	Platysmal banding	<ul style="list-style-type: none"> 7.5 to 20 units per side Electromyographic (EMG) guidance recommended to avoid diffusion to anterior neck <ul style="list-style-type: none"> Strap muscles leads to dysphagia Cricothyroid muscles leads to dysphonia

Botulinum Toxin Complications

	Cause
Lid ptosis	<ul style="list-style-type: none">• Diffusion of botulinum toxin within orbital rim may result in levator paresis; avoid injecting within 1 cm of orbital rim• 0.5% apraclonidine transiently elevates lid margin 1 to 2 mm by activating Müller muscle
Surprised look	Overelevation of medial brow results from excessive weakening of the depressor supercilii portion of corrugator
Dr Spock look	Simultaneous paresis of glabellar complex and preservation of lateral frontalis
Immunoresponse	<ul style="list-style-type: none">• Antibodies can neutralize effects of botulinum toxin; switching serotypes (from botulinum toxin A to botulinum toxin B) ensures chemodenervation• Avoid immunoresponse by injecting smallest dose possible at longest interval possible

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Index

Page numbers followed by “f” indicate figures; those followed by “t” indicate tables; those followed by “b” indicate boxes

A

- ABG. *See* Arterial blood gases.
Abscess, 107
Acellular collagen sponge, 131
Acid-base disorders
 arterial blood gases, 17
 metabolic acidosis, 17–18
 metabolic alkalosis, 17, 19
 respiratory acidosis, 17, 19
 respiratory alkalosis, 17, 20
 venous blood gases, 17
Acidosis
 chronic renal failure as cause of, 14
 metabolic, 17–18
 respiratory, 17, 19
Acinic cell adenocarcinoma, 231, 231f
Acquired melanocytic nevus, 240
Acral lentiginous melanoma, 245, 245f
Actinic keratosis, 242, 242f
Actinomycosis, 112
Acute coronary syndrome, 2
Acute pain, 319
Acute pulmonary diseases, 11–12
Acute renal failure, 13
Acute respiratory distress syndrome, 11, 45
Acute suppurative osteomyelitis, 264
Addison disease, 30
Adenocarcinoma, 231
Adenoid cystic carcinoma, 230, 230f
Adenoidectomy, 157
Adenomatoid odontogenic tumor, 198, 198f
Adipofascial flap, 293
Adrenal disorders, 30, 30f
Advanced trauma life support, 160
Advancement flaps, 125, 125f, 280, 299, 302, 367, 367f
AFO. *See* Ameloblastic fibro-odontoma.
Aggressive fibromatosis, 213–214
Airway
 compromise of, 67
 Mallampati classification of, 50
 preanesthesia evaluation of, 50
 trauma-related complications of, 193
ALARA radiation principle, 77
Albuminuria, 14
Alcohol withdrawal, 39–40
Alkalosis
 metabolic, 17, 19
 respiratory, 17, 20
Allergy, 34
Allodynia, 319
Allografts, 131
All-on-four implant, 128
All-on-six implant, 128
Alveolar cleft bone grafting, 370–371
Alveolar fracture, 173
Alveolar hyperventilation, 20
Alveolar hypoventilation, 19
Alveolar osteitis, 97–98
Alveolar ridge
 augmentation of, 131
 sinus elevation, 132
Alzheimer disease, 40
Ameloblastic fibroma, 200
Ameloblastic fibro-odontoma, 200–201
Ameloblastoma
 benign, 197, 197f
 metastasizing, 202
Ameloblastoma carcinoma, 202–203
Amides, 60
Amoxicillin, 46, 109
Ampicillin, 46, 109
Amyloidosis, 16
Analgesia, 319
Anaphylactic reaction, hypotension with, 64
ANB angle, 140
Ancient schwannoma, 211
Anemia
 chronic renal failure as cause of, 14
 hemolytic, 26
 macrocytic, 25
 microcytic, 25
 nonhemolytic, 26
 normocytic, 25
Anesthesia
 airway evaluation before, 50
 ASA classification, 50
 for asthmatic patient, 72
 barbiturates for, 55–56
 benzodiazepines for, 57–58
 for cerebral palsy patients, 73
 for children, 68
 delayed awakening from, 67
 for diabetic patients, 70–71
 for elderly, 69
 fasting before, 50
 general, 51–52
 for hyperthyroid patients, 71
 intubation techniques used in, 52
 ketamine for, 57
 local anesthetics for, 60–61, 170, 415
 mechanical ventilation, 61–63
 for myasthenia gravis patient, 72
 for neurologic trauma patients, 72
 neuromuscular blockers for, 58
 nondepolarizing agents for, 59

- for obese patients, 69–70
- opioids for, 55
- for pediatric patients, 68
- perioperative management for, 63–66
- postoperative management for, 66–68
- propofol for, 56
- for special populations, 68–73[
- techniques for, 51–52
- in trauma patients, 166–167
- volatile agents for, 53–54, 72
- Anesthesia dolorosa, 319
- Aneurysmal bone cyst, 207
- Angina
 - perioperative management of, 44, 64
 - unstable, 2
- Angiotensin receptor blockers, 6
- Angiotensin-converting enzyme inhibitors, 6
- Angle fracture, 175
- Animal bites, 171, 193
- Anion gap, 18
- Anisocoria, 163
- Ankylosing spondylitis, 342
- Ankylosis, temporomandibular joint, 350–351
- Annulus of Zinn, 184
- Anterior ethmoid artery, 405
- Anterior iliac bone graft, 309–310, 311f, 312
- Anterior plagiocephaly, 382, 382f
- Anterolateral thigh flap, 292–293
- Antibiotics
 - odontogenic infections treated with, 109–110
 - prophylaxis use of, 46
- Anticholinergics, 11
- Anticoagulation
 - contraindications for, 12
 - medications for, 27
 - pulmonary embolus treated with, 12
 - regimens for, 290
- Anti-dsDNA, 36
- Antihelical fold, 409
- Antineutrophil cytoplasmic antibodies, 36
- Antinuclear antibody, 36
- Anti-SSA, 36
- Anti-SSB, 36
- Antoni A/B, in histology, 270
- Antral pseudocyst, 222
- Aortic regurgitation, 3
- Aortic stenosis, 3
- AOT. *See* Adenomatoid odontogenic tumor.
- Apert syndrome, 385–387
- Aphthous stomatitis, 257
- Apicoectomy, 116–117, 117f
- Apnea, 155
- Apnea hypopnea index, 155
- Arch screws, 169
- ARDS. *See* Acute respiratory distress syndrome.
- Arrhythmias, 3–4
- Arterial blood gases, 17–18
- Arthritis
 - infectious, 343
 - osteoarthritis, 33, 339–340
 - psoriatic, 342
 - reactive, 343
 - rheumatoid, 32–33, 339, 341–342
 - temporomandibular joint, 339–344
- Arthrocentesis, 347
- Arthroscopy, 347–348
- Articaine, 60
- Articular cartilage, 332
- Articular disc, 333
- Articular eminence, 332
- ASA classification, 50
- Assist control ventilation, 61
- Asthma
 - anesthesia considerations in, 72
 - characteristics of, 10–11
 - perioperative management of, 45
- Ataxic cerebral palsy, 73
- Atelectasis, 315
- Atracurium, 59
- Atrial fibrillation
 - characteristics of, 4
 - perioperative management of, 44
 - stroke risk assessment in, 4
- Atrial flutter, 4
- Atrophic mandible, 176
- Atrophic maxilla, 106, 127
- Atropine, 68
- Attrition theory, 80
- Augmentin, 109
- Auricular defects, 303
- Autogenous bone grafts, 130
- Autogenous nerve grafts, 97
- Autoimmune diseases
 - myasthenia gravis, 33, 72
 - osteoarthritis, 33
 - rheumatoid arthritis, 32–33
 - sarcoidosis, 32
 - sclerosing syndromes, 34
 - serology tests for, 36
 - systemic lupus erythematosus, 33
 - vasculitides, 35
- Autologous fat transfer, 416
- Avascular necrosis, 147
- Avulsion, 173
- Axial flap, 279
- Axonotmesis, 94

B

- Barbiturates, 55–56
- Bardach two flap technique, 369
- Basal cell adenoma, 228, 228f
- Basal cell carcinoma, 242–244, 244f, 248
- Basilar skull fracture, 164
- Battle sign, 164
- Behçet disease, 35
- Benzodiazepines
 - for anesthesia, 57–58
 - dosages of, 58
- Beta-2 agonists, 10
- Beta blockers
 - for hypertension, 6
 - perioperative use of, 44
- Beta-globulin, 26
- Biguanides, 28
- Bilevel positive airway pressure, 62
- Bilobed flap, 280, 281f, 299
- Binder syndrome, 381
- BiPAP. *See* Bilevel positive airway pressure.

Bipedicled flaps, 302, 302f, 369
 Birbeck granules, in histology, 208f, 270
 BIS. *See* Bispectral index.
 Bispectral index, 51
 Bisphosphonates, 266
 Bites, animal, 171, 193
 Bleeding
 hematologic, 26
 intracranial, 38
 third molar extraction as cause of, 88
 Blepharochalasis, 398
 Blepharoplasty, 400–401
 Blood pressure, 51
 Blood transfusion, 66
 “Blue bloater,” 11
 BMI. *See* Body mass index.
 Body mass index, 69
 Bone grafts/grafting
 alveolar crest, 370–371
 anterior iliac, 309–310, 311f, 312
 cleft lip and palate repaired with, 366, 370–371
 costochondral, 314–315
 from cranial bone, 300
 distant, 309
 iliac crest, 366
 for implant site development, 130–131
 maxillofacial reconstruction using, 308–315
 posterior iliac, 309, 311–312
 regional, 309
 tibial, 309, 312–313
 Bone morphogenetic protein, 371
 Bone plate fixation, 168–169
 Bone quality, 122
 Bone scintigraphy, 263, 337
 Botulinum toxin, 419–420
 Bowen disease, 272
 Brachycephaly, 382, 382f
 Bradycardia
 description of, 4
 hypertension with, 63
 hypotension with, 64
 perioperative management of, 64
 Brain natriuretic peptides, 2
 Branchial arch syndromes
 Binder syndrome, 381
 classification systems, 376
 Goldenhar syndrome, 349, 378
 hemifacial microsomia, 349, 377
 Mobius syndrome, 380
 Nager syndrome, 379
 OMENS classification, 376
 Pierre Robin sequence, 380
 Treacher-Collins syndrome, 349, 378–379
 Branchial cleft cyst, 250–251
 Breslow depth thickness, of melanoma, 246, 246f
 Bronchospasm, 65
 Brow
 ideal position of, 394
 lift technique for, 396
 nasal tip and, esthetic line between, 404
 Buccal space, 109
 Bulbar conjunctiva, 399
 Bupivacaine, 60
 Burkitt lymphoma, 217, 217f

C

Café au lait spots, 269
 Calcifying cystic odontogenic tumor, 201, 201f
 Calcifying epithelial odontogenic tumor, 198, 198f
 Calcium channel blockers, 6
 Calvarial bone graft, 309
 Canalicular adenoma, 228
 Candidiasis, oral, 112–113
 Canine impaction, 79
 Canker sores. *See* Aphthous stomatitis.
 Capnography, 51
 Capsulorrhaphy, 352
 Carcinoma ex pleomorphic adenoma, 231
 Cardiogenic shock, 41
 Cardiogenic syncope, 36
 Cardiovascular diseases and problems
 acute coronary syndrome, 2
 arrhythmias, 3–4
 CHADS₂ scoring table, 4
 congestive heart failure, 2
 heart block, 5f
 hypertension, 6–7
 infectious endocarditis, 7
 perioperative management of, 43, 63–65
 valvular disease, 3
 Caries, dental, 84
 Carotid body tumor, 252
 Carpenter syndrome, 385, 387
 Cartilage reconstruction, 300
 Cat-scratch disease, 250
 Causalgia, 322
 Cavernous sinus thrombosis, 111
 Cawood and Howell classification, of edentulous ridge, 101
 CBCT. *See* Cone beam computed tomography.
 Cefazolin, 46, 109
 Cellulitis, 107
 Cementoblastoma, 200
 Cemento-osseous dysplasia, 209, 210f
 Central diabetes insipidus, 21
 Central giant cell lesion, 206, 206f
 Central granular cell odontogenic tumor, 200
 Central poststroke pain, 322
 Central sensitization, 319
 CEOT. *See* Calcifying epithelial odontogenic tumor.
 Cephalixin, 109
 Cephalogram, 137, 137f
 Cephalometric analysis
 in obstructive sleep apnea syndrome evaluations, 156
 orthognathic surgery uses of, 138f–140f, 138–142
 Cerebral palsy, 73
 Cerebrospinal fluid leak, 164
 Cerebrovascular accident, 37
 Cervical spine
 evaluation of, 143, 143f
 fracture of, 176
 Cervicofacial necrotizing fasciitis, 111–112 Cervicofascial flap, 285
 Cervicomentale angle, 413
 CHADS₂ scoring table, 4
 Cheek reconstruction, 305
 Chemical peel, 418
 Chemosis, 163
 Chemotherapy, for oral squamous cell carcinoma, 237–239
 Cherubism, 207

- Chest pain, 44
CHF. *See* Congestive heart failure.
Children
 anesthesia considerations for, 68
 cutaneous lesions in, 268
 fractures in, 190
 mandibular ramus osteotomies in, 150
 nonodontogenic tumors in, 267
 odontogenic tumors in, 267
 pathology in, 267–268
 salivary gland diseases in, 268
 soft tissue lesions in, 268
 temporomandibular joint ankylosis in, 351
 trauma in, 189–190
Child-Turcotte-Pugh score, 25
Chimeric flap, 293
Chin, 142
Chin-throat angle, 413
Chondroblastic osteosarcoma, 214, 214f
Chondroma, 214
Chondromalacia, 345, 348
Chondrosarcoma, 216, 357
Chronic bronchitis, 11
Chronic nonsuppurative osteomyelitis, 264
Chronic obstructive pulmonary disease, 11
Chronic pain, 319
Chronic recurrent juvenile parotitis, 226
Chronic renal failure, 13–14
Chronic suppurative osteomyelitis, 264
Churg-Strauss syndrome, 16, 35
Chyle leak, 235
Cicatricial ectropion, 398
Cisatracuronium, 59
CIWA protocol. *See* Clinical Institute Withdrawal Assessment for Alcohol protocol.
Clamshell approach, 310, 311f
Clark level staging, of melanoma, 246, 246f
Class II malocclusion, 143
Class III malocclusion, 143
Clear cells, in histology, 270
Clear cell odontogenic carcinoma, 202–203
Clearance, 53
Cleft lip and palate
 alveolar cleft bone grafting for, 370–371
 autogenous bone grafting for, 366, 370–371
 classification of, 364, 365f
 feeding issues secondary to, 365
 incidence of, 364
 lateral muscle bulging secondary to, 375
 lip revision for, 366
 orthognathic surgery in, 366, 374
 pharyngeal flap for, 366
 rhinoplasty for, 366, 373
 secondary deformities, 375
 surgical correction of, 366–374
 Tessier classification system of, 375, 375f
 velopharyngeal insufficiency in, 372, 374
 vermillion-cutaneous mismatch secondary to, 375
 whistle deformity secondary to, 375
Clindamycin, 46
Clinical Institute Withdrawal Assessment for Alcohol protocol, 39
Cluster headache, 324
CMV. *See* Continuous mandatory ventilation.
COD. *See* Cemento-osseous dysplasia.
Codman triangle, 215, 215f, 271
Cold sores. *See* Herpes labialis.
Colloid therapy, 43
Combination syndrome, 101, 101f, 127
Complex focal seizures, 37
Complex regional pain syndrome, 322
Composite free flaps, 295–298
Computed tomographic angiography, 12, 166
Conchal cartilage, 300
Conchal setback, 409
Conchomastoid angle, 408f
Concussion, 172
Condyle, mandibular
 aplasia of, 349
 fracture of, 175, 190
 hypoplasia of, 349
 idiopathic resorption of, 349–350
 unilateral hyperplasia of, 348
Cone beam computed tomography
 implants, 123
 tooth impaction evaluations, 77–78
 trauma evaluations, 166
Congenital heart disease, 46
Congenital hemangioma, 259–260
Congestive heart failure, 2
Conjunctiva, 399
Contact osteogenesis, 124
Continuous mandatory ventilation, 61
Continuous positive airway pressure, 62, 157
COPD. *See* Chronic obstructive pulmonary disease.
Coronal incision, 186
Coronectomy, 92, 92f
Corrugator supercilii, 394, 419
Corticosteroids
 for asthma, 10, 72
 perioperative management of, 46
 for trismus, 98
Cosmetic surgery
 botulinum toxin for, 419–420
 facial analysis for, 392–393
 fillers used in, 419
 forehead, 394–397
 laser resurfacing, 419
 lower third. *See* Lower third of face, esthetic surgery of.
 middle third. *See* Middle third of face, esthetic surgery of.
 skin rejuvenation, 418
 upper third, 394–397
Costal cartilage, 300
Costochondral graft, 314–315
Coxsackie virus, 257, 257f
CPAP. *See* Continuous positive airway pressure.
Cranial nerves
 assessment of, 161
 injury to, 360
Craniofacial disorders
 branchial arch syndromes. *See* Branchial arch syndromes.
 cleft lip and palate. *See* Cleft lip and palate.
 craniosynostosis. *See* Craniosynostosis.
 encephalocele, 388–389
 frontonasal dysplasia, 389
 holoprosencephaly, 388
Craniosynostosis
 characteristics of, 381
 complications of, if left untreated, 383
 definition of, 381

isolated, 382
surgical correction of, 383
syndromic, 385–388
Cribiform adenoid cystic carcinoma, 230, 230f
Cricothyrotomy, 52
Crohn disease, 23
Crouzon syndrome, 385–386
CRPS. *See* Complex regional pain syndrome.
Cryoprecipitate, 66
Crystal-induced arthropathy, 343–344
Crystalloid therapy, 43
Cushing syndrome, 30
Cutaneous lesions
dermatofibrosarcoma protuberans, 247
keratoacanthoma, 247, 247f
lentigo, 240
Merkel cell carcinoma, 247
Mohs surgery for, 247–248
nevus, 240–241
precancerous, 242, 242f
skin cancer, 242–243
Cutis laxa, 277
Cutler-Beard flap, 304, 304f
Cystic teratoma, 251

D

Dautrey procedure, 352
D-dimer test, 12
Dedo facial profile classification, 393
Deep sedation, 51–52
Delanian radiation-induced fibroatrophic theory, of
osteoradionecrosis, 265
Delayed awakening from anesthesia, 67
Delirium, 38
Dementia
characteristics of, 38
Lewy body, 40
Dental arch, 143
Dental caries, 84
Dental implants. *See* Implant(s).
Dental models, 137
Dentigerous cyst, 220
Dentoalveolar injuries, 172–173
Dentoalveolar surgery
apicoectomy, 116–117, 117f
impacted teeth. *See* Third molar impaction; Tooth
impaction.
infections. *See* Infections.
preprosthetic procedures. *See* Preprosthetic surgery.
temporary anchorage devices, 114f, 114–116
Denture-related pathology, 106–107
Depressor supercilii, 394
Dermal melanocytoma, 241
Dermatochalasis, 398
Dermatofibrosarcoma protuberans, 247
Dermis, 418
Dermoid cyst, 251
Desflurane, 54
Desmoplastic fibroma, 213–214
Developmental cysts, 220–222
Dexmedetomidine, 58
Diabetes mellitus
anesthesia considerations in, 70–71

description of, 16, 28–29
perioperative management of, 46
Diabetic ketoacidosis, 29
Dialysis infections, 14
Diastolic blood pressure, 6
Diastolic heart failure, 2
Diazepam, 57–58
DIC. *See* Disseminated intravascular coagulation.
Diplopia, 163, 187
Direct thrombin inhibitors, 27
Disseminated intravascular coagulation, 27
Distance osteogenesis, 124
Distilled water, 118
Distributive shock, 41
Donepezil, 40
Double jaw surgery, 153
Downs analyses, 140, 140f, 142
Downs/Tweed analysis, 141–142
Drainage of infections, 109
Ductal papilloma, 228
Dysesthesia, 319
Dyskinetic cerebral palsy, 73
Dysplastic melanocytic nevi, 242, 242f

E

Ear
lobe deformity, 415
otoplasty of, 408f, 408–410
reconstruction of, 301–303
surface anatomy of, 408, 408f
telephone deformity, 410
trauma to, 164, 172
Ecchymosis, 99, 163, 165
Ectropion, 186, 398
Edentulous patients
implants in, 126–128
mandible, 101
EGFR inhibitor. *See* Epidermal growth factor receptor
inhibitor.
Ehlers-Danlos syndrome, 277
Elderly, anesthesia for, 69
Electrocardiography, 51
Electrolyte disorders
potassium, 22–23
sodium, 20–21
Embolus, pulmonary, 12, 45
Emergency medications, 68
Eminectomy, 352
Emphysema, 11, 99
Encephalocele, 388–389
End stage liver disease, 24–25
Endocarditis
infectious, 7
subacute bacterial, 16
Endocrine diseases
adrenal disorders, 30, 30f
diabetes mellitus, 16, 28–29
diabetic ketoacidosis, 29
pituitary disorders, 30–31
thyroid disorders, 29
Endoneurium, 93
Endoscopic browlift technique, 397

Endotracheal tube
 emergency medications administered via, 68
 intubation using, 52
 for pediatric patients, 68
Enophthalmus posttraumatic, 187
Entropion, 187
Entry wound, 191
Entubulation, 97
Envelope flap, 100
Eosinophilic granuloma, 208
Eosinophilic polygonal cell, in histology, 270
Epidermal growth factor receptor inhibitor, 239
Epidermal nevus, 241
Epidermis, 418
Epidermolysis bullosa, 256
Epidural hematoma, 38
Epilepsy, 45
Epinephrine, 68
Epineurium, 93
Epistaxis, 164
Epithelial odontogenic tumors, 196–198, 197f–198f
Epithelial rests of Serres, 196, 220
Epithelialized palatal graft, 129, 129f
Epithelial-myoepithelial carcinoma, 231
Epulis fissuratum, 106
Erosive lichen planus, 232
ERV. *See* Expiratory reserve volume.
Erythema multiforme, 255, 255f
Erythematous candidiasis, 112
Erythroplakia, 232
ESLD. *See* End stage liver disease.
Esmolol, 63
Esters, 60
Esthetic surgery
 lower third. *See* Lower third of face, esthetic surgery of.
 middle third. *See* Middle third of face, esthetic surgery of.
 upper third, 394–397
Euvolemic hypernatremia, 21
Euvolemic hyponatremia, 21
Ewing sarcoma, 215
Exit wound, 191
Expiratory reserve volume, 9
External carotid artery, 405
Extraparenchymal restrictive diseases, 9
Extra-sinus technique, 128f
Extrusion, of tooth, 173
Extubation, 63, 167
Eyelids. *See also* Lower eyelids; Upper eyelids.
 anatomy of, 182, 398–399
 botulinum toxin complications of, 420
 ptosis of, 420
 reconstruction of, 304, 305f

F

Face
 biologic healing properties of, 170
 thirds of, 138
Facelift, 411–415
Facial analysis, 392–393
Facial angle, 140
Facial artery, 405
Facial artery myomucosal flap, 284, 300

Facial nerve
 description of, 412
 injury to, 171, 360
 mandibular branch of, 412
 temporal branch of, 335, 394, 412
Facial type, 142
Factor 8 deficiency, 26
Factor 9 deficiency, 26
Factor V Leiden disease, 27
Factor Xa inhibitors, 27
Fascial planes, 396f
Fasting, preoperative, 50
FEV1. *See* Forced expiratory volume in 1 second.
Fever, postoperative, 42
Fibroma
 ameloblastic, 200
 desmoplastic fibroma, 213–214
 odontogenic, 199–200
 ossifying, 210
Fibromyalgia, 34, 328
Fibro-osseous disease, 204
Fibro-osseous lesions, 209–210, 210f
Fibrosarcoma, 216, 216f
Fibrous dysplasia, 209, 210f
Fibular flap, 295f, 295–297
Fillers, for cosmetic surgery, 419
Firearm injuries, 191–193
First-degree heart block, 5f
First-pass hepatic effect, 53
Fish-net pattern, in histology, 269
Fitzpatrick sun-reactive skin types, 392
Fixation, for traumatic injuries, 168–169, 175
Fixed porcelain-fused-to-metal bridge, 126
Fixed prosthesis, implant-supported, 126
Flaps
 advancement, 125, 125f, 280, 299, 302, 367, 367f
 anterolateral thigh, 292–293
 bilobed, 280, 281f, 299
 bipedicled, 302, 302f, 369
 cervicofascial, 285
 chest, 287–288
 classification of, 279
 Cutler-Beard, 304, 304f
 envelope, 100
 facial artery myomucosal, 284, 300
 fibular, 295f, 295–297
 glabella, 299
 head, 282f, 282–283
 healing of, to platysma, 417
 helical advancement, 302
 iliac crest, 297–298
 intraoral, 100
 lateral (arm), 294
 lateral (denture), 125, 125f
 latissimus dorsi, 294
 local, 279–281, 280f–281f
 midcrestal incision, 125
 Millard advancement-rotation, 367f–368f, 367–368
 miter, 299
 nasolabial, 283, 299
 neck, 285
 paramedian, 283, 299
 pectoralis major myocutaneous, 287
 pharyngeal, 366, 372
 platysmal, 285

radial forearm fasciocutaneous, 290–291
rectus, 294
regional, 282f, 282–288, 287f
reverse cutback incision, 125
revolving donor island, 303, 303f
rotation, 280, 280f
scapular, 297–298
semilunar, 100
septal mucoperichondrial, 300
sternocleidomastoid, 285
supraclavicular, 286, 287f
temporalis, 282f, 283
temporoparietal, 282, 282f
Tenzel, 304f
transposition, 280, 281f
trapezius, 286
tubed bipedicle postauricular, 302, 302f
ulnar, 294
V to Y advancement, 299, 369
Floor-of-mouth lowering, 105
Fluids
 maintenance requirements for, 66
 overload of, 43
 perioperative management of, 43, 66
Flumazenil, 58
Focal segmental glomerulosclerosis, 15
Focal seizures, 37
Follicular ameloblastoma, 197, 197f
Fondaparinux, 27
Fontaine sign, 269
Forced expiratory volume in 1 second, 9
Forced vital capacity, 9
Forehead surgery, 394–397
Foreign object aspiration, 89
Fractures
 basilar skull, 164
 cervical spine, 176
 in children, 190
 frontal sinus, 180–182
 Le Fort, 177–178
 malunion of, 176
 mandibular, 85, 91, 173–176, 190
 maxillary, 177–178
 nasal, 164, 178, 190
 naso-orbito-ethmoid, 180
 nonunion of, 176, 193
 orbital, 182–187, 190
 panfacial, 187–188
 pediatric, 190
 root, 86
 symphysis, 175
 tuberosity, 90
 zygomaticomaxillary complex, 178–179, 190
Frankfort mandibular plane angle, 142
FRC. *See* Functional residual capacity.
Free flaps
 anterolateral thigh flap, 292–293
 composite, 295–298
 failure of, 289–290
 monitoring of, 289
 radial forearm fasciocutaneous flap, 290–291
 recipient vessels for, 288
 regional flaps versus, 288
 soft tissue, 290–294
Free gingival graft, 129

Frenectomy, 102–103, 103f
Fresh frozen plasma, 66
Frontal bone fractures, 190
Frontal sinus fractures, 180–182, 190
Frontalis, 394, 419
Frontobasilar injuries, 190
Frontonasal dysplasia, 389
Fujita classification, 156
Functional capacity assessment, 8b
Functional residual capacity, 9
Furlow double opposing Z-plasty technique, for cleft palate
 repair, 369, 369f
FVC. *See* Forced vital capacity.

G

Gamma-aminobutyric acid, 39
Gardner syndrome, 213, 272
Garré osteomyelitis, 265
Garrington sign, 215, 215f, 271
Gastroesophageal reflux disease, 24
Gastrointestinal diseases
 end stage liver disease, 24–25
 gastroesophageal reflux disease, 24
 hepatitis, 24
 irritable bowel syndrome, 23
General anesthesia, 51–52
Genioplasty, 152–153
Germectomy, 84
Ghost cells, in histology, 201, 201f, 270
Ghost cell odontogenic carcinoma, 203
Giant cell arteritis, 35
Giant cell lesions, 204–207
Gingival biotype, 123
Gingival cysts, 220
Gingivostomatitis, herpetic, 256
Ginglymoarthrodial joint, 332
Glabella, 404
Glabella flap, 299
Glandular odontogenic cyst, 220, 221f
Glasgow Coma Scale, 160
Glogau photoaging classification, 392
Glomerular filtration rate, 14
Glomerulonephritis, 16
Glossopharyngeal nerve, 409
Glossopharyngeal neuralgia, 321
Glyburide, 28
Goiter, 251
Goldenhar syndrome, 349, 378
Goode method, 403
Goodpasture syndrome, 15
Gorlin cyst, 201, 201f
Gorlin syndrome, 272
Gout, 343–344
Granular ameloblastoma, 197
Gray, 78
Great auricular nerve, 97, 409, 412
Guillain-Barré syndrome, 45

H

Hallermann-Streiff syndrome, 349
Halo nevus, 241

- Hand-foot-mouth disease, 257, 257f
 - Handgun injuries, 191–192
 - Hand-Schuller-Christian disease, 208, 271
 - Hashimoto thyroiditis, 251
 - Head
 - fascial planes of, 396f
 - firearm injuries to, 191–193
 - flaps of, 282f, 282–283
 - imaging of, in oral squamous cell carcinoma, 233
 - Headaches, 323–325
 - Heart block, 5f
 - Heerfordt syndrome, 272
 - Helical advancement flap, 302
 - Hemangioma, 258–260
 - Hematologic diseases
 - anemia, 25–26
 - bleeding disorders, 26
 - disseminated intravascular coagulation, 27
 - hypercoagulable state, 27
 - sickle cell disease, 26
 - Hematoma, 38, 99, 165, 186, 401, 410, 415
 - Hematopoietic stem cell transplantation, 232
 - Hemifacial microsomia, 349, 377
 - Hemimandibular elongation, 348
 - Hemimandibular hyperplasia, 348
 - Hemoglobin A1c, 28–29
 - Hemolytic anemia, 26
 - Hemophilia A, 26
 - Hemophilia B, 26
 - Hemorrhage, 42, 133, 162
 - Hemorrhagic stroke, 37
 - Hemostatic agents, 88
 - Hemotympanum, 164
 - Heparin, 27
 - Heparin-induced thrombocytopenia, 27
 - Hepatitis, 24
 - Herpangina, 257
 - Herpes labialis, 256
 - Herpes simplex virus, 256
 - Herpes zoster virus, 257
 - Herpetic gingivostomatitis, 256
 - Herringbone pattern, in histology, 216, 216f, 270
 - Hertel exophthalmometer, 162
 - Hertwig root sheath, 196, 198
 - Heterotopic bone formation, 361
 - Heterotopic pain, 319
 - Holdaway ratio, 142
 - Holoprosencephaly, 388
 - Hughes procedure, 304, 305f
 - Huntington disease, 40
 - Hurler syndrome, 349
 - Hutchinson sign, 269
 - Hydralazine, 63
 - Hyperalgesia, 319
 - Hypercalcemia, 31
 - Hypercoagulable state, 27
 - Hyperglycemia, 29, 71
 - Hyperkalemia
 - characteristics of, 22–23
 - chronic renal failure as cause of, 14
 - Hyponatremia, 21
 - Hyperparathyroidism, 205
 - Hyperpituitarism, 31
 - Hyperplastic candidiasis, 112
 - Hypersensitivity reactions, 34
 - Hypertension
 - with bradycardia, 63
 - characteristics of, 6–7
 - perioperative management of, 43, 63
 - with tachycardia, 63
 - Hypertensive emergencies, 7
 - Hyperthermia, malignant, 65
 - Hyperthyroidism
 - anesthesia considerations in, 71
 - characteristics of, 29
 - Hypertonic hyponatremia, 20
 - Hypertrophic scar, 278
 - Hypervolemic hypernatremia, 21
 - Hypervolemic hyponatremia, 21
 - Hyphema, 163
 - Hypoalgesia, 319
 - Hypocalcemia, 31
 - Hypoglycemia, 71
 - Hypoglycemics, oral, 28
 - Hypokalemia, 22
 - Hyponatremia, 20–21
 - Hypopituitarism, 31
 - Hypopnea, 155
 - Hypotension, 64
 - Hypothyroidism, 29
 - Hypotonic hyponatremia, 20
 - Hypoventilation
 - obesity hypoventilation syndrome, 69
 - postoperative, 67
 - Hypovolemic hypernatremia, 21
 - Hypovolemic hyponatremia, 20
 - Hypovolemic shock, 41
- I**
- Idiopathic condylar resorption, 153, 349–350
 - Idiopathic trigeminal neuropathic pain, 321
 - IE. *See* Infective endocarditis.
 - IgA nephropathy, 16
 - Iliac crest bone graft, 366
 - Iliac crest flap, 297–298
 - Immediate implant, 129
 - Immune complex glomerulonephritis, 16
 - Immune thrombocytopenic purpura, 26
 - Immunoresistance, 420
 - Impacted teeth. *See* Third molar impaction; Tooth impaction.
 - Implant(s)
 - all-on-four, 128
 - all-on-six, 128
 - bone grafting for, 130–131
 - complications of, 133
 - components of, 120–121
 - crest module design of, 120–121, 121f
 - in edentulous patients, 126–128
 - failure of, 133
 - fixed prosthesis supported with, 126
 - imaging of, 123
 - immediate, 129
 - nonsubmerged approach to, 125
 - one- versus two-stage approach to, 125
 - osseointegration of, 124
 - placement of, 124
 - presurgical workup for, 122–123
 - prosthetic complications of, 133

restorative space requirements for, 122, 123f
ridge augmentation for, 131
sinus elevation for, 132
site development for, 129–132
socket preservation, 131–132
soft tissue augmentation for, 129–130
space requirements for, 122, 123f
submerged approach to, 125
surface of, 120
surgical principles for, 124–125
uncovering of, 125
zygomatic, 128
Implant-retained, tissue-supported prosthesis, 127
Incisions
 coronal, 186
 infraorbital, 185
 Killian, 406
 lateral brow, 186
 for orbital fracture repair, 185–186
 postauricular, 336
 preauricular, 336
 retromandibular, 336
 subciliary, 185
 submandibular, 336
 transcaruncle, 186
 transconjunctival, 185
 transfixion, 406
 transmaxillary/transnasal, 185
 vertical releasing, 100
Incisor mandibular plane angle, 141
Indian-file pattern, in histology, 270
Infantile hemangioma, 258–259
Infections
 dialysis, 14
 drainage of, 109
 liposuction-related, 417
 odontogenic, 107–110
 osteoplasty-related, 410
 rhinoplasty-related, 406
 after temporomandibular joint surgery, 360
 types of, 110–113
 vesiculobullous, 256–257
 wound, 99
Infectious arthritis, 343
Infective endocarditis, 7
Inferior alveolar nerve injury, 92, 97, 151
Inflammatory odontogenic cysts, 219
Inflammatory papillary hyperplasia, 107
Inflammatory salivary gland disorders, 224–226
Infraorbital incision, 185
Infratemporal fossa, 87
Infratemporal space, tooth/root displacement in, 87
Instruments, broken, 89
Insulin, 28, 46
Insulin glargine, 28
Insulin Lispro, 28
Intercondylar width, 151
Internal carotid artery, 405
Interstitial emphysema, 99
Intracranial bleeding, 38
Intracranial hypertension, 383
Intraoral flaps, 100
Intrusion, of tooth, 173
Intubation
 endotracheal, 52

 pediatric, 68
 in trauma patients, 166
Inverted-L osteotomy, 148–152
Irritable bowel syndrome, 23
Ischemic stroke, 37
Isoflurane, 54
Isotonic hyponatremia, 20

J

Jacobson nerve, 409
James Brown classification, of midface defects, 307, 307f
Jaw
 medication-related osteonecrosis of, 266–267
 metastatic carcinoma of, 218
 tumors of, 267
Juvenile ossifying fibroma, 210, 210f
Juvenile rheumatoid arthritis, 342

K

Kaban protocol, 351, 377
Kasabach-Merritt syndrome, 272
Kazanjian flap vestibuloplasty, 104, 104f
Keloids, 278, 410
Kenalog injection, for scars, 281
Keratoacanthoma, 247, 247f
Ketamine, 57
Ketoacidosis, diabetic, 29
Kidney failure. *See* Renal failure.
Kiesselbach plexus, 405
Killian incision, 406

L

Labial frenectomy, 102, 103f
Lacrimal duct injury, 172
Lag screw fixation, 168
Lagophthalmos, 401
Lambdoid synostosis, 384, 384f
Langerhans cell disease, 204
Langerhans cell histiocytosis, 207, 208f
Laryngeal mask airway, 52, 72
Laryngospasm, 65
Laser resurfacing, 419
Lateral arm flap, 294
Lateral brow incision, 186
Lateral canthal angle, 398
Lateral canthus, 183
Lateral check ligament, 183
Lateral flap advancement, 125, 125f
Lateral luxation, 173
Lateral periodontal cyst, 220
Lateral pharyngeal space, 109
Lateral pterygoid myotomy, 352
Latissimus dorsi flap, 294
Le Fort fractures, 177–178
Le Fort I osteotomy, 144–147
Lentigo maligna melanoma, 245, 245f
Leser-Trélat sign, 269
Lesser occipital nerve, 409
Letterer-Siwe disease, 208, 272

Leukoplakia, 232
Leukotriene modifiers, for asthma, 10
Levator palpebrae superioris muscle, 399
Lewy body dementia, 40
Lichen planus, oral, 253f–254f, 253–254
Lidocaine, 60, 68
Lidocaine toxicity, 417
Liesegang rings, 198, 198f, 269
Ligaments, ocular, 183
Limberg rhomboid flap, 280, 281f
Lingual frenectomy, 103
Lingual nerve injury, 93, 97
Lip(s)
 cleft. *See* Cleft lip and palate.
 injury to, 172
 reconstruction of, 306
Lipodystrophy, 416
Liposuction, 416–417
5-Lipoxygenase, for asthma, 11
“Lip-switch” vestibuloplasty, 105, 105f
LMA. *See* Laryngeal mask airway.
Load-bearing, 169
Load-sharing, 169
Local anesthetics/anesthesia, 60–61, 170, 415
Local flaps, 279–281, 280f–281f
Lockwood ligament, 193
Long buccal nerve injury, 93
Lorazepam, 57–58
Lower eyelids
 anatomy of, 182
 blepharoplasty of, 400
 retraction of, 401
 transconjunctival blepharoplasty of, 400
Lower lip reconstruction, 306
Lower third of face
 anatomy of, 138
 esthetic surgery of
 facelift, 411–415
 liposuction, 416–417
Low-molecular-weight heparin, 27
Lung volumes, 9f
Lymph node metastasis, 236
Lymphangioma, 250
Lymphoepithelial cyst, 223

M

MAC. *See* Minimum alveolar concentration.
Macrocytic anemia, 25
Magnetic resonance imaging, 337
Main sensory nucleus, 318
Malignant fibrous histiocytoma, 217
Malignant hyperthermia, 65
Malignant peripheral nerve sheath tumor, 216–217
Mallampati classification, of airway, 50
Malocclusion, 165, 361
Malunion, of fracture, 176
Mandible
 anteroposterior position of, 140, 140f
 atrophic, 176
 edentulous, 101
 fracture of, 85, 91, 173–176, 190
 reconstruction of, 308
Mandibular canal, tooth/root displacement in, 86

Mandibular incisors
 crowding of, 83
 inclination analysis of, 141
Mandibular nerve, 318
Mandibular plane angle, 141–142
Mandibular ramus
 bone grafts from, 309
 osteotomy of, 148–152
Margin reflex distance, 398
Marie-Strumpell disease, 342
Marjolin ulcer, 272
Marx theory, of osteoradionecrosis, 265
Masticatory muscle disorders, 325–328
Mathes and Nahai system, for muscular flap classification, 279
Maxilla
 anteroposterior position of, 138, 138f
 atrophy of, 127f
 fractures of, 177–178
Maxillary artery, 405
Maxillary incisor inclination analysis, 141
Maxillary nerve, 318
Maxillary sinus, tooth/root displacement in, 86
Maxillary submucosal vestibuloplasty, 104
Maxillary surgery, 144–147
Maxillary torus palatinus, 106
Maxillary tuberosity
 fracture of, 90
 surgical reduction of, 102
Maxillofacial complex trauma, 165
Maxillofacial reconstruction
 autogenous bone grafts/grafting for, 308–315
 cheek, 305
 ear, 301–303
 eyelid, 304, 305f
 free flaps. *See* Free flaps.
 lip, 306
 local flaps, 279–281, 280f–281f
 mandible, 308
 midface, 307–308
 nose, 298–301
 regional flaps, 282f, 282–288, 287f
 wound healing, 276–278
Maxillomandibular advancement, 158
Maxillomandibular fixation screws, 169
McCune-Albright syndrome, 271
McGregor patch, 413
McNamara analysis, 138–141, 139f
Mechanical ventilation, 61–63
Mechanoreceptors, 93, 95
Medial canthus, 183
Medial check ligament, 183
Medications
 anticoagulation, 27
 cardiovascular, 6, 8
 endotracheal tube administration of, 68
 for hypertension, 6
 opioids, 55
 osteonecrosis of the jaws caused by, 266–267
 pharmacokinetics of, 53
 surgical management of patients on, 8
 volatile, 53–54, 72
Meglitinides, 28
Melanocytic nevus, 240–241
Melanoma, 244–246, 245f–246f
Melanotic neuroectodermal tumor of infancy, 212

Melkersson-Rosenthal syndrome, 272
 Melolabial crease, 413
 Membranoproliferative glomerulonephritis, 16
 Membranous nephropathy, 15
 MEN. *See* Multiple endocrine neoplasia.
 Mepivacaine, 60
 Merkel cell carcinoma, 247
 Mesencephalic nucleus, 318
 Mesenchymal odontogenic tumors, 196, 199f–200f, 199–200
 Metabolic acidosis, 17–18
 Metabolic alkalosis, 17, 19
 Metabolic equivalents of task, 8b
 Metastatic carcinoma of the jaws, 218
 Metformin, 28
 Methohexital, 55–56
 Meyer theory, of osteoradionecrosis, 265
 Microcytic anemia, 25
 Microscopic polyangiitis, 35
 Microsurgery, 95
 Midazolam, 57–58
 Midcrestal incision flap, 125
 Middle third of face
 anatomy of, 137
 esthetic surgery of
 blepharoplasty, 400–401
 otoplasty, 408f, 408–410
 periorbital anatomy, 397–399
 rhinoplasty, 401–407
 Midface
 fractures of, 190
 reconstruction of, 307–308
 Midline palatal cyst, 222
 Migraine headache, 323
 Millard advancement-rotation flap, 367, 367f
 modified, 368, 368f
 Mini-facelift, 414
 Minimal change disease, 15
 Minimal sedation, 51–52
 Minimum alveolar concentration, 53
 Miniplates, 114f, 114–115
 Miniscrews, 114f, 114–115
 Miter flap, 299
 Mitral regurgitation, 3
 Mitral stenosis, 3
 Mitral valve prolapse, 3
 Mitral valve prolapse syndrome, 3
 MMA. *See* Maxillomandibular advancement.
 Mobius syndrome, 380
 Moderate sedation, 51–52
 Modified Aldrete discharge criteria, 67
 Modified radical neck dissection, 235
 Modified Wells criteria, 12
 Mohs surgery, 247–248
 Monomorphic adenoma, 227
 MPNST. *See* Malignant peripheral nerve sheath tumor.
 Mucocoele, 223
 Mucoepidermoid carcinoma, 229, 229f–230f
 Mucormycosis, 113
 Mucous retention cyst, 222
 Mueller muscle, 399
 Multinodular goiter, 251
 Multiple endocrine neoplasia, 212
 Multiple myeloma, 16, 218, 218f
 Muscle contracture, 326–327
 Muscle spasm, 326–327

Myasthenia gravis, 33, 72
 Myasthenic crisis, 33
 Mylohyoid nerve injury, 93
 Myocardial infarction, 2
 Myoclonic seizures, 37
 Myoepithelioma, 228
 Myofascial pain, 326–328
 Myositis, 326–327
 Myxoma, odontogenic, 199, 199f

N

Nager syndrome, 379
 Naloxone, 55, 68
 Nasal fractures
 in adults, 164, 178
 in children, 190
 Nasal intubation, in trauma patients, 166
 Nasal lining reconstruction, 300
 Nasal obstruction, 407
 Nasal reconstruction, 298–301
 Nasal septum, 403, 407
 Nasal structures, 402–403
 Nasal tip, 402–403, 407
 Nasolabial flap, 283, 299
 Naso-orbito-ethmoid fractures, 180
 Nasopalatine duct cyst, 222
 Native valve endocarditis, 7
 Nausea and vomiting, postoperative, 67
 Neck
 dissection of, 234–236
 fascial planes of, 396f
 firearm injuries to, 191–193
 flaps of, 285
 imaging of, in oral squamous cell carcinoma, 233
 lymph node metastasis in, 236
 masses of, 248–252
 metastatic rate for, 236
 trauma to, 165
 Necrotizing fasciitis, cervicofacial, 111–112
 Neoplasms, temporomandibular joint, 353–357
 Neostigmine, 58
 Nephritic disease, 16–17
 Nephrotic disease, 15–16, 18
 Nephrotic syndrome, 15–16
 Nerve(s)
 mapping chart for, 95, 96f
 terminology associated with, 94
 Nerve fiber, 93
 Nerve injury
 from facelift, 415
 from third molar extraction
 autogenous grafts for, 97
 classification of, 94
 inferior alveolar nerve, 92, 97
 lingual nerve, 93, 97
 long buccal nerve, 93
 mylohyoid nerve, 93
 repair of, 95–97
 Neurilemmoma, 211
 Neurofibroma, 211
 Neurogenic syncope, 36
 Neurogenic tumors, 204, 211–214, 212f
 Neuroleptic malignant syndrome, 38–39

Neurologic disorders
 alcohol withdrawal, 39–40
 Alzheimer disease, 40
 cerebrovascular accident, 37
 declined mental ability, 38
 Huntington disease, 40
 intracranial bleeding, 38
 Lewy body dementia, 40
 neuroleptic malignant syndrome, 38–39
 Parkinson disease, 40
 seizure, 37
 serotonin syndrome, 39
 stroke, 37
 syncope, 36, 65
Neurologic trauma, 72
Neuromuscular blockers, 58
Neuropathic pain disorders, 320–322
Neuropaxia, 94
Neurorrhaphy, 95
Neurotropism, 269
Nevus, 240–241
Nikolsky sign, 255, 269
Nitrous oxide, 54
Nociceptors, 93, 95
Nodular melanoma, 245, 245f
Noma, 113
Nondepolarizing agents, 59
Nonhemolytic anemia, 26
Nonodontogenic tumors
 benign, 204–214
 fibro-osseous lesions, 209–210, 210f
 giant cell lesions, 204–206
 Langerhans cell histiocytosis, 207, 208f
 malignant, 214f–218f, 214–218
 neurogenic tumors, 204, 211–214, 212f
 overview of, 204
 pediatric, 267
Non-ST segment elevation myocardial infarction, 2
Nonsuppurative osteomyelitis, 264–265
Nonunion, of fracture, 176, 193
Normocytic anemia, 25
Nose
 blood supply of, 405
 inferior turbinate of, 403
 obstruction of, 407
 reconstruction of, 298–301
 rhinoplasty of. *See* Rhinoplasty.
 septum of, 403
 structures of, 402–403
 surface anatomy of, 404
 tip of, 402–403, 407
NPH Insulin, 28
NSTEMI. *See* Non-ST segment elevation myocardial infarction.

O

Obesity, 69–70
Obesity hypoventilation syndrome, 69
Obstructive lung diseases, 9–10
Obstructive sleep apnea, 69
Obstructive sleep apnea syndrome, 155–158
Oculo-auriculo-vertebral dysplasia, 376
Oculoauriculovertebral syndrome, 349
Oculomandibulodyscephaly, 349

Odontogenic carcinoma, 202–203
Odontogenic cysts
 developmental, 220–222
 inflammatory, 219
 overview of, 219
Odontogenic fibroma, 199
Odontogenic infections, 107–110
Odontogenic keratocyst, 221, 221f
Odontogenic myxoma, 199, 199f
Odontogenic tumors. *See also specific tumor.*
 benign, 196–201, 203
 epithelial, 196–198, 197f–198f
 malignant, 196, 202–203
 mesenchymal, 196, 199f–200f, 199–200
 mixed, 196, 200–202, 201f
 origins of, 196
 pediatric, 267
 prognosis for, 203
 types of, 196
Odontoma, 201
Oncocytoma, 228, 228f
Open reduction and internal fixation, 175
Ophthalmic artery, 405
Ophthalmic nerve, 318
Opioids, 55
Oral candidiasis, 112–113
Oral intubation, in trauma patients, 166
Oral lichen planus, 253f–254f, 253–254
Orbicularis oculi, 394, 399, 419
Orbit, 184
Orbital apex syndrome, 163
Orbital fractures
 in adults, 182–187
 in children, 190
Orbital hematoma, 186
Orbital ligament, 395
Orbital septum, 399
Oroantral communication, 90–91
Orofacial arterial system, 283–284
Orofacial clefting, 375, 375f. *See also* Cleft lip and palate.
Orofacial gangrene, 113
Orofacial pain
 acute, 319
 chronic, 319
 headaches, 323–325
 masticatory muscle disorders, 325–328
 neuroanatomy and neurophysiology of, 318–319
 neuropathic pain disorders, 320–322
 terminology associated with, 319
Orthodontics, before orthognathic surgery, 143
Orthognathic surgery, 83
 in cleft lip and palate patients, 366, 374
 controversies in, 154
 genioplasty, 152–153
 indications for, 136
 Le Fort I osteotomy, 144–147
 maxillary, 144–147
 obstructive sleep apnea syndrome treated with, 155–158
 stability of, 153
 surgical-assisted rapid palatal expansion, 145
 treatment planning for
 cephalometric analysis, 138f–140f, 138–142
 clinical work-up, 136–137
 facial examination, 137–138
 growth evaluation, 143
 presurgical orthodontics, 143
 surgical work-up, 137

Orthostatic syncope, 36
OSAS. *See* Obstructive sleep apnea syndrome.
Osseointegration, 124
Ossifying fibroma, 210
Osteoarthritis, 33
Osteoblastoma, 204, 212–213, 355
Osteochondroma, 353–354
Osteoconduction, 124, 130
Osteocutaneous flap, 291
Osteogenesis, 124, 130
Osteoid osteoma, 204, 212–213, 355
Osteoinduction, 130
Osteoma, 213, 355
Osteomyelitis, 176, 193, 262–265
Osteonecrosis of the jaws, medication-related, 266–267
Osteoradionecrosis, 265–266
Osteosarcoma, 214f, 214–215, 356
Osteotomy
 inverted-L, 148–152
 Le Fort I, 144–147
 mandibular ramus, 148–152
 rhinoplasty, 406
 sagittal split, 148–152
 sandwich, 131
 vertical ramus, 148–152
Otoplasty, 408f, 408–410
Otorrhea, 164
Overdenture, implant-supported, 127
Oxidized cellulose plant polymer, 88

P

Pain
 central poststroke, 322
 chest, 44
 heterotopic, 319
 myofascial, 326–328
 orofacial. *See* Orofacial pain.
 referred, 319
 third molar extraction-related, 97–98
Palatal graft, epithelialized, 129, 129f
Palatal island flap, 284
Palatal roll technique, 130, 130f
Palate, cleft. *See* Cleft lip and palate.
Palpebral conjunctiva, 399
Palpebral fissure, 398
Pancuronium, 59
Panfacial fractures, 187–188
Panoramic radiographs, 166
PAP. *See* Pulmonary artery pressure.
Papillary cystadenoma lymphomatosum, 227, 227f
Paradental cyst, 219
Paralytics, 72
Paramedian flap, 283, 299
Paresthesia, 319
Parkinson disease, 40
Parotid duct injury, 172, 415
Paroxysmal hemicrania, 324
Paroxysmal supraventricular tachycardia, 4
Partial specific volume, 62
Pathology
 correlations for, 269–272
 cutaneous lesions. *See* Cutaneous lesions.
 medication-related osteonecrosis of the jaws, 266–267
 neck masses, 248–252
 nonodontogenic tumors. *See* Nonodontogenic tumors.
 odontogenic tumors. *See* Odontogenic tumors.
 oral squamous cell carcinoma. *See* Squamous cell carcinoma, oral.
 osteomyelitis, 176, 193, 262–265
 osteoradionecrosis, 265–266
 pediatric, 267–268
 radiologic correlations for, 271
 thyroid masses, 251–252
 vascular anomalies, 258–262
 vesiculobullous diseases, 253f–255f, 253–257
Pauci immune glomerulonephritis, 16
PE. *See* Pulmonary embolus.
Pectoralis major myocutaneous flap, 287
Pediatrics. *See* Children.
Pedicle flap from palate, 130, 130f
PEEP. *See* Positive end-expiratory pressure.
Pell and Gregory classification system, for third molar impaction, 81f
Pemphigoid, 253f, 253–254
Pemphigus, 253f, 253–254
Penetrating wounds, 191
Penicillin, 109
Pentobarbital, 55–56
Perforating wounds, 191
Perforator flap, 279
Periapical cyst, 219
Pericarditis, 14
Pericoronitis, 83
Peri-implantitis, 133
Perineurium, 93
Periodontal defects, 99
Periodontal disease, 84
Perioperative management
 acute respiratory distress syndrome, 45
 angina, 44, 64
 antibiotic prophylaxis, 46
 asthma, 45
 atrial fibrillation, 44
 bradycardia, 64
 bronchospasm, 65
 cardiovascular diseases, 43, 63–65
 corticosteroids, 46
 diabetes mellitus, 46
 epilepsy, 45
 fever, 42
 fluid management, 43, 66
 hemorrhage, 42
 hypertension, 44
 hypotension, 64
 issues for, 43–46
 laryngospasm, 65
 malignant hyperthermia, 65
 monitoring tools, 51
 pulmonary embolus, 45
 shock, 41–42
 status epilepticus, 45
 syncope, 65
 tachycardia, 64
Periorbital region, 397–399
Peripheral sensitization, 319
Perivascular hyalinosis, in histology, 270
Permanent cavity, 191
Pfeiffer syndrome, 385, 387
PHACE syndrome, 259, 272

Pharmacokinetics
 description of, 53
 in elderly, 69
Pharmacology
 opioids, 55
 pharmacokinetics, 53
 volatile agents, 53–54, 72
Pharyngeal flap, 366, 372
Pickwickian syndrome, 69
Pierre Robin sequence, 380
Pigmented villonodular synovitis, 353–354
Pilomatrixoma, 268
Pindborg tumor, 198, 198f
“Pink puffer,” 11
PIOC. *See* Primary intraosseous odontogenic carcinoma.
Pioglitazone, 28
Pituitary disorders, 30–31
Plagiocephaly, 382, 382f, 384, 384f
Plasma osmolality, 20
Plate fixation, 168–169
Platelets, 66
Platinum-based chemotherapy agents, 238
Platysma, 419
Platysmal flap, 285
Pleomorphic adenoma, 227, 227f
Pleomorphic undifferentiated sarcoma, 217
Pleural lacerations, 315
Plexiform ameloblastoma, 197, 197f
Plexiform neurofibroma, 212f
Pneumatization, 132
Pneumonia, 45
Pneumothorax, 315
Polly-beak deformity, 406
Polyarteritis nodosa, 35
Polymorphous low-grade adenocarcinoma, 231
Polysomnography, 155
Porcelain-fused-to-metal bridge, 126
Port-wine stain, 269
Positional plagiocephaly, 384, 384f
Positive end-expiratory pressure, 62
Postauricular incision, 336
Posterior auricular artery, 409
Posterior iliac bone graft, 309, 311–312
Postherpetic neuralgia, 322
Postoperative management
 anesthesia, 66–68
 fever, 42
 nausea and vomiting, 67
 wound infection, 99
Postradiation sarcoma, 218
Poststreptococcal glomerulonephritis, 16
Posttraumatic headache, 325
Potassium disorders, 22–23
Pradaxa, 27
Preauricular incision, 336
Premolar impaction, 79
Preprosthetic surgery
 edentulous mandible, 101
 frenectomy, 102–103, 103f
 tuberoplasty, 105
 tuberosity reduction, 102
 vestibuloplasty, 103–105, 104f–105f
Pressure-controlled ventilation, 61
Presurgical orthodontics, 143
Prilocaine, 60

Primary adrenal insufficiency, 30
Primary generalized seizures, 37
Primary intraosseous odontogenic carcinoma, 202
Primary nephrotic syndrome, 15
Primordial cyst, 220
Procerus, 394, 419
Progressive systemic sclerosis, 34
Proliferative periostitis, 265
Proliferative verrucous leukoplakia, 232
Prominauris, 408
Propofol, 56
Proptosis, 163
Prosthetic valve endocarditis, 7
Protein C deficiency, 27
Protein S deficiency, 27
Pseudo-ankylosis, 351
Pseudogout, 343–344
Pseudomembranous candidiasis, 112
Pseudomembranous colitis, 110
Psoriatic arthritis, 342
PSV. *See* Partial specific volume.
PSVT. *See* Paroxysmal supraventricular tachycardia.
Pterygomandibular space, 109
Pulmonary artery pressure, 53
Pulmonary embolus, 12, 45
Pulse oximetry, 51
Pupillary reflexes, abnormal, 163
PVS. *See* Pigmented villonodular synovitis.
Pyrimidine analog, 238

R

Radial forearm fasciocutaneous flap, 290–291
Radiation dose, 78
Radiation therapy
 adjuvant therapy and, 238
 oral squamous cell carcinoma treated with, 236–237
Radical neck dissection, 235
Radiographs
 tooth impaction localization using, 77
 trauma examination using, 166
Radix, 404
Random flap, 279
Ranula, 223
Rapid palatal expansion, surgical-assisted, 145
Reactive arthritis, 343
Reconstruction
 maxillofacial. *See* Maxillofacial reconstruction.
 orbital, 186
 for panfacial fractures, 188
 temporomandibular joint, 358–359
Rectus flap, 294
Red blood cells, 66
Redistribution, 53
Referred pain, 319
Reflex sympathetic dystrophy, 322
Regional flaps, 282f, 282–288, 287f
Reiter syndrome, 343
Renal disease
 acute renal failure, 13
 chronic renal failure, 13–14
 nephrotic disease, 15–16
Renal failure
 acute, 13
 chronic, 13–14

Renal osteodystrophy, 14
Repaglinide, 28
Residual volume, 9
Respiratory acidosis, 17, 19
Respiratory alkalosis, 17, 20
Respiratory diseases and problems
 acute, 11–12
 acute respiratory distress syndrome, 11, 45
 asthma, 10–11, 45
 chronic, 10–11
 chronic obstructive pulmonary disease, 11
 lung volumes, 9f
 pulmonary embolus, 12, 45
 types of, 9
Respiratory disturbance index, 155
Restrictive lung diseases, 9–10
Rests of Malassez, 196
Rests of Serres, 196, 220
Retrolbulbar hematoma, 163
Retromandibular incision, 336
Retropharyngeal space, 109
Reverse cutback incision flap, 125
Revolving donor island flap, 303, 303f
Rheumatoid arthritis, 32–33, 339, 341–342
Rheumatoid factor, 36
Rhinon, 404
Rhinoplasty
 anatomy of, 401–405
 cleft, 366, 373
 complications of, 406–407
 osteotomy for, 406
 techniques for, 405–406
Rickets analysis, 138–139
Ridge augmentation, 131
Rifle injuries, 191–192
Rigid fixation, 169
Rocker deformity, 407
Rocuronium, 59
Root displacement, 86–87
Root fracture, 86
Root resorption, 85
Rotation flap, 280, 280f
RV. *See* Residual volume.

S

Saddle-nose deformity, 407
Saethre-Chotzen syndrome, 385, 388
Sagittal split osteotomy, 148–152
Salivary gland diseases
 cystic lesions, 223
 inflammatory, 224–226
 non-neoplastic, 223–231
 obstructive, 226
 pediatric, 268
Salivary gland tumors
 benign, 227–228
 malignant, 229f–231f, 229–231
 overview of, 226–227
Sandwich osteotomy, 131
SAPHO syndrome. *See* Synovitis acne pustulosis hyperostosis
 osteitis syndrome.
Sarcoidosis, 32, 225
Sarcoma
 chondrosarcoma, 216, 357
 Ewing, 215
 fibrosarcoma, 216, 216f
 osteosarcoma, 214f, 214–215, 356
 pleomorphic undifferentiated, 217
 postradiation, 218
 synovial, 357
Scalp, 162, 394
Scaphocephaly, 382, 382f
Scapular flap, 297–298
Scars, 278, 281
Schwannoma, 211, 212f
Scintigraphy, 263, 337
Scleroderma, 34
Sclerosing osteomyelitis, 264–265
Sclerosing syndromes, 34
Screw fixation, 168–169
Seborrheic keratosis, 241
Second molar impaction, 79
Secondary hypertension, 6
Secondary nephrotic syndrome, 16
Second-degree heart block, 5f
Sedation
 description of, 51–52
 for obese patients, 69
Seddon classification, of nerve injuries, 94
Seizure, 37
Selective neck dissection, 235
Semilunar flap, 100
Sensitization, 319
Sentinel vein, 394
Septal cartilage, 300
Septal hematoma, 164
Septal mucoperichondrial flap, 300
Serology tests, 36
Serotonin syndrome, 39
Sevoflurane, 54
Shingles, 257
Shock, 41–42
Shotgun injuries, 191–192
Sialadenitis, 224–225
Sialolithiasis, 226
Sickle cell disease, 26
Sievert, 78
Simon method, 403
Simple focal seizures, 37
Simple lentigo, 240
SIMV. *See* Synchronized intermittent mandatory ventilation.
Sinus elevation, 132
Sipple syndrome, 272
Sjögren syndrome, 34, 225
Skin cancer
 basal cell carcinoma, 242–244, 244f, 248
 melanoma, 244–246, 245f–246f
 nonmelanoma, 242–244, 244f
 squamous cell carcinoma, 242–244, 244f
Skin evaluation, 392
Skin rejuvenation, 418
Skin slough and necrosis, from facelift, 415
Sleep endoscopy, 156
SNA angle, 138, 138f
SNB angle, 140
Socket preservation, 131–132
Sodium bicarbonate, for metabolic acidosis, 18

- Sodium disorders, 20–21
- Soft tissue
 - augmentation of, for implant placement, 129–130
 - free flaps, 290–294
 - injuries to, 170–172
- Solar lentigo, 240
- SOT. *See* Squamous odontogenic tumor.
- Spastic cerebral palsy, 73
- Sphincter pharyngoplasty, 372
- Spinal trigeminal nucleus, 318
- Spitz nevus, 241
- Spondyloarthropathies, 342–343
- Squamous cell carcinoma
 - cutaneous, 242–244, 244f
 - oral
 - adjuvant treatment for, 236–239
 - chemotherapy for, 237–239
 - epidemiology of, 232
 - head and neck imaging for, 233
 - neck dissection for, 234–236
 - radiation therapy for, 236–237
 - TNM classification of, 232
 - verrucous carcinoma, 239
- Squamous odontogenic tumor, 198, 198f
- ST segment elevation myocardial infarction, 2
- Starry-sky pattern, in histology, 270
- Status epilepticus, 37, 45
- Steiner analyses, 138f, 138–142
- STEMI. *See* ST segment elevation myocardial infarction.
- Sternocleidomastoid flap, 285
- Still disease, 343
- Stroke
 - hemorrhagic, 37
 - ischemic, 37
 - risk assessment for, 4–5
- Sturge-Weber syndrome, 262, 272
- Subacute bacterial endocarditis, 16
- Subarachnoid hematoma, 38
- Subciliary incision, 185
- Subdural hematoma, 38
- Subepithelial connective tissue graft, 130, 130f
- Sublingual space, 109
- Subluxation
 - dentoalveolar, 172
 - temporomandibular joint, 352
- Submandibular incision, 336
- Submandibular space
 - drainage of infection in, 109
 - tooth/root displacement in, 86
- Submarginal flap, 100
- Submental intubation, in trauma patients, 166
- Submental island flap, 284
- Submental space, 109
- Submucous fibrosis, 232
- Subperiosteal abscess, 99
- Succinylcholine, 58–59
- Sulfonylureas, 28
- Summer technique, 132
- Sunderland classification, of nerve injuries, 94
- “Sun-ray” appearance, of osteosarcoma, 214, 214f
- Superficial muscular aponeurotic system, 402, 413–414
- Superficial spreading melanoma, 245, 245f
- Superficial temporal artery, 409
- Superior orbital fissure syndrome, 163
- Superior orbital fractures, 190
- Suppurative osteomyelitis, 264
- Supraclavicular flap, 286, 287f
- Supraorbital nerve, 394
- Supratrochlear nerve, 394
- Sural nerve, 97
- Surgery
 - airway evaluation before, 50
 - cardiac risk factors, 8b
 - cosmetic. *See* Cosmetic surgery.
 - dentoalveolar. *See* Dentoalveolar surgery.
 - fasting before, 50
 - orthognathic. *See* Orthognathic surgery.
 - in patients on cardiovascular medications, 8
 - perioperative monitoring, 51
 - preprosthetic. *See* Preprosthetic surgery.
 - risk levels of, 8b
- Surgical-assisted rapid palatal expansion, 145
- Suture resorption time, 117
- Swiss-cheese pattern, in histology, 270
- Symphysis
 - bone grafts from, 309
 - fracture of, 175
- Synchronized intermittent mandatory ventilation, 62
- Syncope
 - characteristics of, 36
 - perioperative management of, 65
- Syndromic craniosynostosis, 385–388
- Synovial cavity, 333
- Synovial chondromatosis, 354
- Synovial fluid, 333
- Synovial sarcoma, 357
- Synovitis, 345, 348
- Synovitis acne pustulosis hyperostosis osteitis syndrome, 265
- Systemic lupus erythematosus, 33
- Systolic blood pressure, 6
- Systolic heart failure, 2

T

- Tachycardia
 - hypertension with, 63
 - hypotension with, 64
 - paroxysmal supraventricular, 4
 - perioperative management of, 64
 - ventricular, 3
- Takayasu arteritis, 35
- Target therapy, 239
- Tarsal plates, 399
- Taxane chemotherapy agents, 238
- Technetium-99m bone scan, 263, 348
- Telecanthus, traumatic, 163
- Telephone ear deformity, 410
- Temporal arteritis, 324
- Temporal artery, 409
- Temporalis flap, 282f, 283
- Temporary anchorage devices, 114f, 114–116
- Temporomandibular joint
 - alloplastic reconstruction of, 358–359
 - anatomy of, 332–334
 - ankylosis of, 350–351
 - arthritis of, 339–344
 - arthrocentesis of, 347
 - arthroscopy of, 347–348
 - benign tumors of, 355

- capsule of, 335
- chondromalacia of, 345, 348
- clicking of, 346
- components of, 332–334
- contusion of, 175
- crystal-induced arthropathy of, 343–344
- dislocation of, 352–353
- disorders of, 338
- growth disorders of, 348–350
- hypermobility of, 352–353
- hypomobility of, 350–351
- imaging of, 337
- incision designs for, 336
- internal derangements of, 345–348
- ligaments of, 334
- locking of, 346
- magnetic resonance imaging of, 337
- malignant neoplasms of, 356–357
- musculature associated with, 334
- neoplasms of, 353–357
- pain of, 98
- prostheses, 358–359
- pseudotumors of, 354
- reconstruction of, 358–359
- spondyloarthropathies, 342–343
- subluxation of, 352
- surgery of, 359–361
- surgical anatomy of, 335–336
- surgical entrance to, 335–336
- synovitis of, 345, 348
- unilateral condylar hyperplasia, 348
- Temporoparietal flap, 282, 282f
- Teninocutaneous flap, 291
- Tennison-Randall procedure, 367
- Tenon capsule, 184
- Tension-type headache, 323
- Tenzel flap, 304f
- Tessier approach, 310, 311f
- Tessier classification system, of orofacial clefting, 375, 375f
- Tetanus immunization, 171
- Thiazide diuretics, 6
- Thiazolidinediones, 28
- Third molar extraction
 - complications of
 - iatrogenic damage, 89–93
 - intraoperative, 86–89
 - nerve injury, 92–97
 - postoperative, 97–99
 - contraindications to, 85
 - indications for, 83–85
- Third molar impaction
 - ADA codes for, 82
 - classification systems, 81–82
 - extraction for. *See* Third molar extraction.
 - management of, 80–99
 - mandible fracture risks, 85
 - mandibular, 81
 - maxillary, 81
 - Pell and Gregory classification systems, 81f
 - reasons for, 80
 - Winter classification system, 82, 82f
- Third-degree heart block, 5f
- Thoracic scoliosis, 315
- Thrombin, 88
- Thrombophilias, 27
- Thyroglossal duct cyst, 251
- Thyroid disorders, 29
- Thyroid masses, 251–252
- Tibial bone graft, 309, 312–313
- Tidal volume, 9
- Tissue engineering, 131
- TLC. *See* Total lung capacity.
- TNM classification, 232
- Tongue flap, 284
- Tonic-clonic seizures, 37
- Tonsillectomy, 157
- Tooth impaction
 - canines, 79
 - chart for, 76f
 - cone beam computed tomography of, 77–78
 - definition of, 76
 - etiology of, 79
 - factors associated with, 77
 - frequency of, 76, 79
 - location determinations for, 77–78
 - management of, 79
 - premolars, 79
 - radiographic evaluations, 77
 - second molars, 79
 - third molars. *See* Third molar impaction.
- Torus mandibularis, 106
- Total lung capacity, 9
- Tracheostomy, 52, 167
- Tracheotomy, 167
- Transcaruncle incision, 186
- Transconjunctival blepharoplasty, 400
- Transconjunctival incision, 185
- Transfixion incision, 406
- Transforming growth factor, 131
- Transmaxillary/transnasal incision, 185
- Transposition flap, 280, 281f
- Transpositional flap “lip-switch” vestibuloplasty, 105, 105f
- Transverse cervical system, 286, 287f
- Transverse nasalis, 419
- Trapdoor approach, 310, 311f
- Trapezius flap, 286
- Trauma
 - advanced trauma life support assessment, 160
 - anesthesia considerations in, 72
 - anesthesia in, 166–167
 - clinical evaluation of, 160–166
 - complications of, 193
 - cranial nerve assessment, 161
 - dentoalveolar, 172–173
 - ear, 164, 172
 - firearm injuries, 191–193
 - fixation principles for, 168–169, 175
 - frontal sinus fractures, 180–182
 - hemorrhage classification, 162
 - mandibular fractures, 173–176, 190
 - maxillary fractures, 177–178
 - maxillofacial complex, 165
 - nasal cavity, 164
 - nasal fractures, 178
 - naso-orbito-ethmoid fractures, 180
 - neck, 165
 - neurologic, 72
 - ocular, 163
 - orbital fractures, 182–187
 - panfacial fractures, 187–188
 - pediatric, 189–190
 - physical examination for, 162
 - radiographic examination for, 166
 - soft tissue injuries, 170–172

Traumatic bone cyst, 222
Treacher-Collins syndrome, 349, 378–379
Trigeminal nerve, 409
Trigeminal neuralgia, 320–321
Trigonocephaly, 382, 382f
Trismus, 98, 351
Triton tumor, 272
Trotter syndrome, 272
Tschopp approach, 310, 311f
Tubed bipediced postauricular flap, 302, 302f
Tuberoplasty, 105
Tubular adenoid cystic carcinoma, 230, 230f
Tumescent liposuction technique, 416
TV. *See* Tidal volume.
Type 1 diabetes mellitus, 28
Type 2 diabetes mellitus, 28

U

Ulcerative colitis, 23
Ulnar flap, 294
Unstable angina, 2
Upper eyelids
 blepharoplasty of, 400
 description of, 182
 retractors of, 399
 skin crease incision, 186
Upper lip reconstruction, 306
Upper third of face
 anatomy of, 137
 esthetic surgery of, 394–397
Urinalysis
 chronic renal failure findings, 15
 nephritic disease findings, 17
 nephrotic disease findings, 17

V

Vagus nerve, 409
Valvular disease, 3
Vascular anomalies, 258–262
Vascular malformation, 258, 260–261
Vasculitides, 35
Vasopressin, 68
Vasovagal syncope, 36
VBG. *See* Venous blood gases.
Vecuronium, 59
Velopharyngeal insufficiency, 372, 374
Venous blood gases, 17
Ventricular tachycardia, 3
Vermilion reconstruction, 306
Vermilion-cutaneous mismatch, 375

Verocay bodies, in histology, 270
Verrucous carcinoma, 239
Vertical ramus osteotomy, 148–152
Vertical releasing incisions, 100
Vesiculobullous diseases, 253f–255f, 253–257
Vestibular schwannoma, 211
Vestibuloplasty, 103–105, 104f–105f
Volatile agents, 53–54, 72
Volume of distribution, 53
Volume-controlled ventilation, 61
Vomiting, 67
Von Langenbeck technique, for cleft palate repair, 369, 369f
Von Recklinghausen disease, 272
von Willebrand disease, 26
von Willebrand factor deficiency, 26
V to Y advancement flap, 299, 369

W

Wallerian degeneration, 93
Warfarin, 27
Warthin tumor, 227, 227f
Wegener granulomatosis, 16, 35
Wermer syndrome, 272
Whistle deformity, 375
Whitnall ligament, 193
Wickham striae, 254f
Winter classification system, for third molars, 82, 82f
Wits analysis, 140–141
Wolff-Parkinson-White syndrome, 4
Wound(s)
 characteristics of, 191
 closure of, 170
 healing of, 170, 276–278
 penetrating, 191
 perforating, 191
Wound infection, postoperative, 99

X

Xenografts, 131

Z

Z-plasty
 description of, 280
 Furlow double opposing, for cleft palate repair, 369, 369f
 labial frenectomy treated with, 102, 103f
Zygoma anatomy-guided approach, 128, 128f
Zygomatic implant, 128
Zygomaticomaxillary complex fractures, 178–179, 190
Zygomycosis, 113